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A JOURNAL OF NEUROLOGY.

VOL. II.



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# BRAIN:

A JOURNAL OF NEUROLOGY.

EDITED BY

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# CONTENTS.



## ORIGINAL ARTICLES:—

PAGE

The Re-Education of the Adult Brain. By William Sharpey, M.D., LL.D., F.R.S. . . . .	1
Observations on Neuritis and Peri-Neuritis of some of the Cranial Nerves. By Julius Althaus, M.D., M.R.C.P. Lond. . . . .	10
Auditory Vertigo. By J. Hughlings-Jackson, M.D., F.R.S. . . . .	29
Note on a Reflex Mechanism in the Fixation of the Eyeballs. By W. R. Gowers, M.D. . . . .	39
On the Weight of the Brain and its Component Parts in the Insane. By J. Crichton-Browne, M.D., LL.D. . . . .	42
Psychometric Experiments. By Francis Galton, F.R.S. . . . .	149
On the Electrical Excitability of the Skin. By S. Tschiriew, M.D. (St. Pet.), &c., and A. de Watteville, M.A. (Lond.), &c. . . . .	163
The Eye-Symptoms in Locomotor Ataxia. By T. Grainger Stewart, M.D., &c. . . . .	181
Remarks on Bulbar Paralysis, with Special Reference to Artificial Feeding. By Thomas Stretch Dowse, M.D., F.R.C.P. (Ed.) . . . . .	191
On Affections of Speech from Disease of the Brain. By J. Hughlings-Jackson, M.D., F.R.C.P., F.R.S. . . . .	203, 323
Vomiting in connection with Cerebral Disease. By D. Ferrier, M.D., F.R.S. . . . .	223
Sunstroke and some of its Sequelæ. By Sir Joseph Fayrer, K.C.S.I., F.R.S. . . . .	297
"Re-Education of the Adult Brain." By J. Mortimer Granville, M.D. . . . .	317
The Therapeutic Value of Citrate of Caffein as a General Sedative, Anodyne, and Diuretic. By Lewis Shapter, M.D. (Cantab.) . . . .	357
Notes on certain Lesions of the Nervous Tissues of frequent occurrence in the Brain of the Insane. By Bevan Lewis, L.R.C.P. . . . .	364
On the Balance of Pressure within the Skull. By James Cappie, M.D. . . . .	373
Chronic Morphinism. By H. Obersteiner, M.D. . . . .	449
Illustrations of the Diseases of the Pons Varolii. By W. R. Gowers, M.D. . . . .	466
On the Origin of Tetanus. By Surgeon-Major J. J. L. Ratton, M.D. . . . .	478
Heredity and Crime in Epileptic Criminals. By Henry Clarke, L.R.C.P. (Lond.) . . . . .	491

## CRITICAL DIGESTS AND NOTICES OF BOOKS:—

	PAGE
Engelmann on the Hystero-Neuroses. By A. Hughes Bennett, M.D. . . . .	68
Barlow on Regressive Paralysis. By Thomas Barlow . . . . .	73
Murphy on Habit and Intelligence. By F. L. Benham, M.B. . . . .	78
Dowse on the Brain and its Diseases. By T. Clifford Allbutt . . . . .	95
Huxley on Hume. By John Charles Bucknill . . . . .	99
Luciani and Tamburini on the Functions of the Brain. The Psycho-sensory Cortical Centres. By A. Rabagliati . . . . .	234
Grant Allen on the Colour Sense: its Origin and Development. By F. L. Benham . . . . .	250
Gowers' Manual and Atlas of Ophthalmoscopy. By T. Clifford Allbutt, M.D. . . . .	385
Lewes on the Study of Psychology. By F. L. Benham, M.B. . . . .	390
Boyer's Etudes Cliniques sur les Lésions Corticales. By D. Ferrier, M.D., F.R.S. . . . .	400
Clifford's Lectures and Essays. By J. C. Bucknill, M.D., F.R.S. . . . .	403
Balfour's Defence of Philosophical Doubt. By J. C. Bucknill, M.D., F.R.S. . . . .	528
Calderwood's Relations of Mind and Brain. By W. W. Ireland, M.D. . . . .	535
Müller on the Acute Anterior Poliomyelitis of the Adult. By A. de Watteville . . . . .	540

## CLINICAL CASES:—

On Simple Aphasia, and Aphasia with Incoherence. By Dr. Magnan . . . . .	112
A Case of Chorea, in which Death was caused by Cerebral Hæmorrhage. By E. Buchanan Baxter, M.D., F.R.C.P. . . . .	124
Some Cases of Nerve Resection and Nerve Stretching. Reported by Herbert W. Page, M.A., F.R.C.S. . . . .	128
Case of Locomotor Ataxy. By C. F. Newcombe, M.D. . . . .	134
A Case of Double Optic Neuritis, with Remarks on the Causation of Optic Neuritis. By Stephen Mackenzie, M.D. . . . .	257
Case of Purulent Cerebral and Spinal Meningitis, &c. By Joseph W. Hunt, M.D. . . . .	409
Clinical Cases of Hernia Cerebri. By John Duncan, M.A., M.D. . . . .	413
Case of Acute Ascending Paralysis. By Arthur W. Fox, M.B. . . . .	418
Puncture of Lens: Ophthalmitis: Brain Disease: Death. By T. Pridgin Teale, M.A., F.R.C.S. . . . .	423
Case of Pseudo-hypertrophic Spinal Paralysis occurring in an Adult. By A. Hughes Bennett, M.D. . . . .	426
Case of Acute Traumatic Tetanus. By J. J. L. Ratton, M.D. . . . .	430



# CONTENTS.

vii

Five Cases of Disease of the Brain, &c. By Charles K. Mills, M.D. . . . .	547
Cases of Intracranial Tumour. By James Ross, M.D. . . . .	569
A Case of Hemiplegia due to Wasting of Cerebral Convolutions. By Francis Warner, M.D., M.R.C.P., and Fletcher Beach, M.B., M.R.C.P. . . . .	576

## ABSTRACTS OF BRITISH AND FOREIGN JOURNALS:—

Progressive Amyotrophic Bulbar Paralysis—A New Theory of Locomotor Ataxia—Progressive Muscular Atrophy—Multiple Cerebro-Spinal Sclerosis—The Influence of the Nervous System on the Irritability of the Motor Nerves. By W. J. Dodds, M.B., D.Sc. . . . .	139
Pathological Lesions versus Motor Centres—Diagnosis of Lesions of the Cortex Cerebri—Ergot in Insanity. By W. W. Ireland, M.D. . . . .	144
The Spinal Ganglia and Spinal Cord of the Petromyzon—Proliferations of the Ependyma Ventriculorum. By H. Obersteiner . . . . .	147
Hystero-Neuroses. By Robert Lawson, M.B. . . . .	148
On Metallo- and Magneto-Therapeutics. By A. de Watteville . . . . .	275
On the Cephalic Ganglion and Retina in the Arthropoda (Berger). On the Structure of a Microcephalic Brain (Rohon) By H. Obersteiner . . . . .	285
On the Termination of Nerves in Striated Muscles. The tone of Striated Muscle (Tschiriew). By D. Ferrier . . . . .	286
Note on Infantile Paralysis. By Thomas Barlow . . . . .	289
Gray on Heredity in Epilepsy. By J. Crichton-Browne . . . . .	290
A Series of Annotations. By A. R. Urquhart . . . . .	291
Case of Unilateral Epilepsy. Una Microcefala. By W. W. Ireland . . . . .	296
Report on Visceral Neurology. By J. Mitchell Bruce, M.D. . . . .	434
Locomotor Ataxy, and its Connection with Injuries (Petit). By Herbert W. Page . . . . .	440
Cases of Bulbar Paralysis (Erb); Conclusions from the Study of 125 Cases of Writers' Cramp and Allied Affections (Beard). By A. de Watteville . . . . .	443
Dimensions of the Fœtal Head (Budin and Ribement). By A. R. Urquhart, M.D. . . . .	445
American Neurological Notes. By R. Lawson, M.B. . . . .	446
On the Sensory Centres of the Cortex Cerebri (Luciani and Tamburini). By W. W. Ireland . . . . .	580
On a Case of Progressive Muscular Atrophy, &c. (Erb and Schultze); Concussion of the Spinal Cord (Obersteiner); Localisation of Atrophic Spinal Paralysis (Remak). By A. de Watteville . . . . .	581
On the Structure of the Cortex Cerebri (Stricker and Unger); Shrivelling and Atrophy of the Cornu Ammonia in Epilepsy; A Case of Cortical Epilepsy. By H. Obersteiner . . . . .	585

	PAGE
On the Pathological Anatomy of Hydrophobia (Weller): On Combined Disease of the Spinal Tracts (Westphal). By W. J. Dodds, M.D., D.Sc. . . . .	586
Head Measurements (Wight). By Crochley Clapham . . . .	591
General Paralysis amongst Negroes. By Robert Lawson, M.B. . . .	592

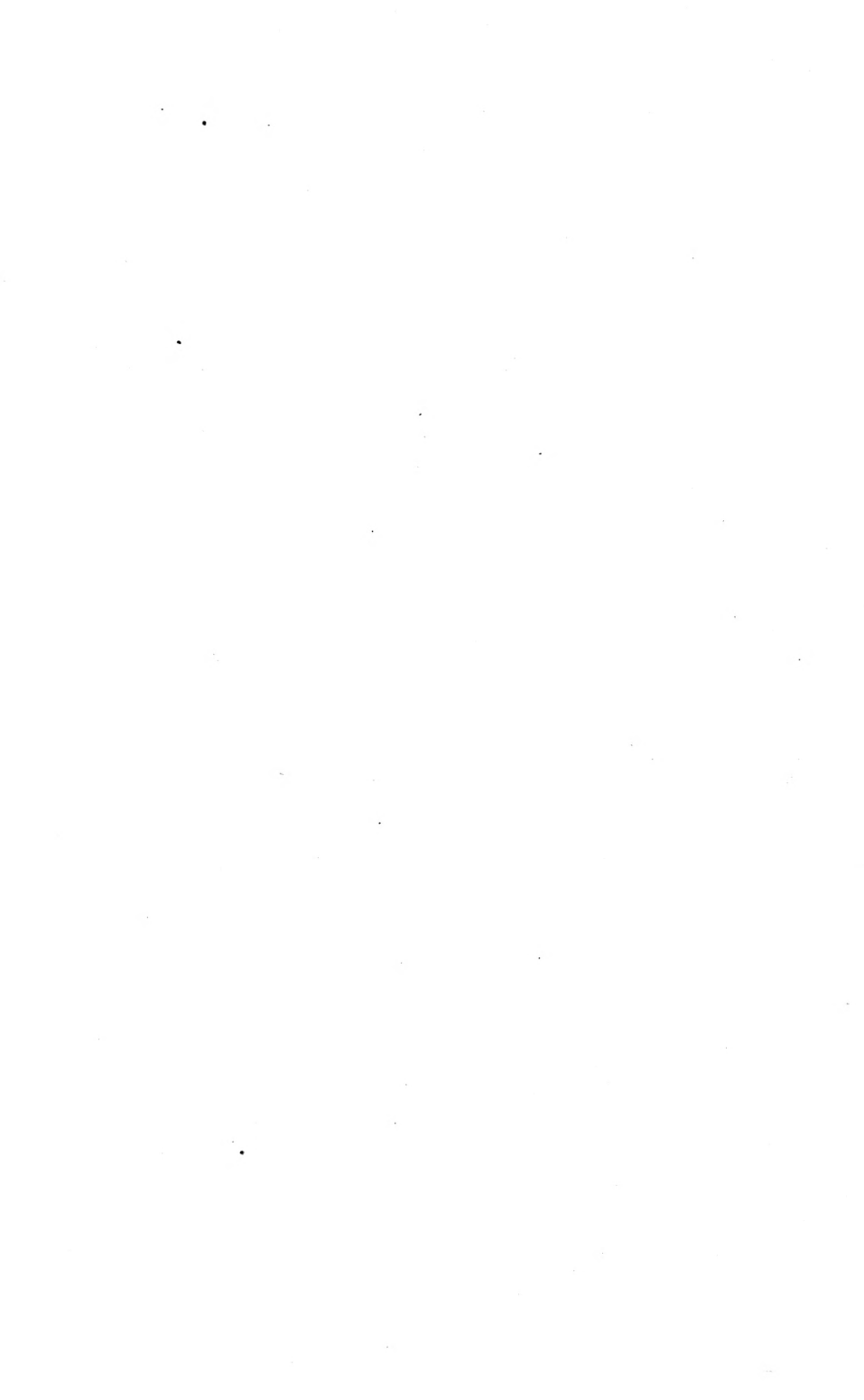
## CORRESPONDENCE:—

Note on Dr. J. Hughlings-Jackson's Case of Auditory Vertigo in April Number of 'BRAIN' . . . . .	273
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WARNER, FRANCIS, M.D.,  
M.R.C.P.



## LIST OF ILLUSTRATIONS.

Aphasia :—	PAGE
Simple Aphasia (Observations 1 and 2) . . . . .	113
Aphasia with Incoherence (Observation 3) . . . . .	117
Sarcoma of Neuroglia . . . . .	121
Indented Cavity of the Left Hemisphere, caused by the Sarcoma of Neuroglia . . . . .	121
Electrode for Testing the Electrical Excitability of the Skin ( <i>Woodcut</i> )	165
Functional Localisations of the Cerebral Cortex of the Dog, according to Munk ( <i>Woodcuts</i> ) . . . . .	236
A Case of Double Optic Neuritis (Section through deeper part of Frontal Convolution, &c.) . . . . .	266
Certain Lesions of Nervous Tissues in the Brain of the Insane . . .	369
Illustrations of Diseases of the Pons Varolii :—	
Section through the Crura Cerebri, just above the Pons ( <i>Woodcut</i> )	473
Section through the Left Half of the Pons Varolii at Level of Exit of Fifth Nerve ( <i>Woodcut</i> ) . . . . .	476
Charcot's Scheme of Optic Tracts ( <i>Woodcut</i> ) . . . . .	574
The Ear in a Case of Hemiplegia due to Wasting of Cerebral Convolution	578



# B R A I N.

APRIL, 1879.

## Original Articles.

### THE RE-EDUCATION OF THE ADULT BRAIN.

BY WILLIAM SHARPEY, M.D., LL.D., F.R.S.,

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MRS. H., the subject of the following case, is about 24 years of age,\* of a pale complexion and slender make. She was married in July 1823, and, with the exception of occasional headaches to which she, in common with some of the rest of her family, was subject, and slight bowel complaints, she previously to that time enjoyed good health, both of body and mind. After her marriage, she resided in England till the end of April 1824, when, in consequence of ill-health, she was brought here by her husband, on a visit to her friends who live in this neighbourhood,† and with whom she had passed a great part of her previous life. From her husband's account, it appeared that for about three months after their marriage she enjoyed perfect health and spirits, but that after that time she complained a good deal of pain in her stomach and bowels; her appetite was bad, she began to lose spirits, imagining herself unequal to the concerns of the house, though her family consisted only of herself and her husband; and now also it was observed that she slept more than usual. The medical gentlemen consulted, believing some of her complaints might be

\* This was written in 1824.

† Arbroath, Forfarshire.

owing to an affection of the liver, administered mercury in small quantities, and applied leeches to the temples in considerable numbers, with a view to relieve an uneasy feeling of lightness which she complained of in her head; but these remedies proved of little or no avail, and for some time before leaving England, excepting a laxative which she took twice a-week, she had given up the use of medicine altogether. No probable cause could be assigned for these complaints.

On her arrival in this place, which, as already mentioned, was about the end of April last, she had lost but little flesh, and by no means looked sickly; indeed she was little, if at all, changed in her appearance; all her external senses were sound, but her memory was impaired, and she was very inattentive to surrounding objects, which made her dull and absent in company. The sleepiness had been very gradually increasing, and was now arrived at such a height that, unless when conversing with another person, or engaged in some manual occupation, she fell asleep at all times, and in whatever situation or position she might be. When in this state her eyes were nearly close, she breathed softly, and, in short, very much resembled a person in natural sleep, except that when she happened to fall asleep in a position in which the body naturally requires to be supported, as for instance on a chair, she did not lean forwards or backwards as is commonly the case, but sat with her body quite erect, and her head gently inclined to one side. While in this state she was subject to frequent startings, during which she raised herself up, talked as if she were frightened, drew herself back as if to avoid something disagreeable, and then after a few seconds lay quietly down again without having awoke. What she said on these occasions, though quite incoherent, was yet always nearly of the same nature, and for the most part consisted even of the same expressions, which were those of great aversion or horror; of this she had no recollection when awake, nor of anything connected with it; and she herself remarked as something extraordinary that now she did not dream, although she used formerly to be very subject to dreaming. From this sleep she never awoke of her own accord, except to obey the calls of nature; and there was no other way of rousing her



up upon other occasions, but by placing her on her feet and endeavouring to make her walk. When thus forcibly awakened, she was fretful, and cried for some time after. She took food in sufficient quantity, and often with evident relish; but it required much entreaty to make her take the first two or three mouthfuls. The pulse varied a little, but on the whole was nearly natural; during sleep it was commonly from 56 to 70, and somewhat more when awake. Her bowels were very costive, and constantly required the use of laxative medicine; the discharge of urine was natural; the catamenia had hitherto been regular in their appearance, but in small quantity. She complained of no pain or other uneasiness, except a peculiar feeling in the top of the head across the bregma, which she called "funny."

For five weeks after her arrival the torpid state and indifference to surrounding objects continued gradually to become worse, and the difficulty of awakening her daily increased; till at length, about the 8th or 10th of June, it was found impossible to rouse her up at all by any means that could be thought of; and from that time forth, excepting a few short intervals, she remained in a state of constant sleep till the beginning of August. Her condition was now singular enough. She still made an attempt to get out of bed when she required to go to stool; when food was presented to her lips with a spoon, she readily took it into her mouth and swallowed it, and in this way she was fed as long as the torpor continued; when she had taken what appeared a sufficiency, she closed her teeth as a sign she was satisfied, and if importuned to take more, turned away her mouth from the spoon. She appeared also to distinguish different tastes, for she gave an evident preference to some sorts of food and obstinately refused others. She sometimes even, to all appearance, judged of the nature of the food or medicines offered to her, by the sense of smelling; and when the latter were such as possessed a strong odour, she would often close her mouth in a determined manner before they touched her lips.

By this time the startings in her sleep had left her; and although the expressions she uttered when in that state were, with some slight additions, nearly the same as formerly, yet

her manner of speaking was now indicative of satisfaction and not of fright. She often even sang to a simple but cheerful air nearly the same words which she used formerly to cry out with every appearance of the greatest terror.

The torpor continued nearly in the same degree till the end of July, with occasional intervals of waking, which happened at uncertain periods, but generally at the distance of a few days from each other, and were occasioned by pain experienced in some part of her body. The first of these took place after she had been ten days in a continued state of torpor; it was caused by severe griping from laxative medicine. She awakened in great suffering, crying out, "pain," "pain;" "die," "die;" and placing her hands on the abdomen. She was relieved by means of warm fomentations; but she, notwithstanding, kept awake for some hours after, during which time she answered no question, in however loud a voice it was put to her, and recognised nobody except one old acquaintance, whom she had not seen for more than twelve months. She looked steadfastly in this person's face for a few seconds, apparently occupied in trying to remember his name, which at length she found out and repeated again and again, at the same time taking him by the hand as if overjoyed to see him; but when questioned regarding him, she answered only by calling out his name, which she continued to repeat for some time after she had fallen asleep, in addition to what she usually said. In the course of the next eight days she was twice roused from her sleep by a similar cause, but not so completely; the same individual was still the only person she knew; amongst others she did not recognise even her own husband, who happened then to be in Scotland.

The next interval of waking took place three or four days afterwards; it appeared to be occasioned by pain in the head. She cried for some time, then awoke, complaining of pain, with her hand on the forepart of her head, on which also she placed the hand of a person near her and pressed it down firmly with her own; after thus complaining for two or three hours, she fell asleep. The same thing happened on the next and the two or three succeeding evenings, nearly at the same hour, but each time with less complaint. Other circumstances

about this time showed that she was suffering considerable uneasiness in her head. She was very impatient in the erect posture, and, when lifted out of bed, would not put her feet to the ground, but drew up her legs to her body, as if to force those who held her to lay her down again. This, however, was not the case when she required to be taken up for the purpose of making any evacuation. She generally also preferred to lie on her face, and always with her head very low, with both hands firmly clasped over it, exactly on the part to which she had formerly referred the peculiar feeling already mentioned, and showed much uneasiness when they were removed, unless the pressure was continued by the hand of another person.

After this, the torpor continued for some time without being interrupted; but in the meantime the symptoms of pain in the head, and the uneasiness in the erect posture, gradually wore off, and Mrs. H. now no longer talked in her sleep. Her bowels were kept open by laxative medicine, which now did not operate so severely as to wake her. She had, since the beginning of June, had a blister applied to the nape of the neck, and three to the head at different periods; sinapisms to the feet were also had recourse to, and two or three times electric shocks were passed through her arms. These remedies, like other painful stimulants, caused her to complain much; and one of the blisters, which was sufficiently large to cover the whole scalp, made her open her eyes; but their effects were merely temporary, leaving, to all appearance, no permanent impression on her complaint. Lest there might be any serous effusion within the cranium, digitalis was used along with the sweet spirit of nitre, in such quantity as greatly to augment the flow of urine. By its operation her pulse was reduced so low as 44 in a minute; and, while using it, she appeared to suffer from sickness at the stomach, during which she often put her fingers into her mouth, as if wishing for something to eat or drink; and she was subject to what seemed an oppressive feeling in the region of the heart, with a peculiar interruption to her breathing, which came in paroxysms; all which symptoms left her after discontinuing the medicine.

Towards the latter end of July, the torpid state, which had suffered no more intermissions, was become on the whole not

quite so deep ; at least Mrs. H. now gave signs of being more conscious of anything that was done to her. She smiled and seemed pleased on receiving particular sorts of food, and when her eye was opened, or any part of her face touched with a finger, her whole countenance became suffused with a blush of redness. Some short time after, it became possible to awake her by opening her eyes, and holding anything before them likely to catch her attention, such as a glass of water, a cup, or the like. When awaked in this way, which succeeded best at the times she was getting her food, she generally laughed a good deal and seemed much delighted, and she always bestowed her whole attention on the vessel in which her food or drink was contained, and the person who held it ; she, however, did not speak, and paid no attention whatever to the questions put to her. One day about this time, viz. on the 1st of August, in consequence of her usual medicines failing in their effect, she had two or three laxative clysters, and then a small dose of croton oil, which produced very copious evacuations, but at the same time caused so much griping as to wake her. When suffering from this, she took hold of the blankets of her bed, twisted them in her hands, and applied them over the abdomen, looking wistfully all the while in the faces of the attendants, as if she had recollected the fomentations which had formerly given her relief, and wished them to be had recourse to on the present occasion ; her wish was complied with, with the effect of removing the pain, which seemed to give her great satisfaction. In two or three days after this the torpor was much diminished, and she could be awakened with great ease. She likewise began to take a great liking to the young woman who waited on her, so much so that, when awake, she would hardly allow her to be a moment out of her sight. Now also she would sometimes let herself cautiously down on the floor from her bed, and creep to the fireside, where she would lay herself quietly down on the hearthrug, as if she wished to enjoy the warmth of the fire.

At length, after progressively improving for some days, she was by the third week in August almost free from torpor, and slept little more than a person in health. During all this period, except that her feet were sometimes cold, the temperature of

her body was very nearly natural. Her face was for the most part pale, but sometimes a little flushed, and the pupil of the eye uniformly contracted on exposure to the light. Her pulse, which had been rendered slow by the digitalis, was observed to be rather higher for some time preceding her recovery than it had been even before the use of that medicine. She had undoubtedly lost flesh during her illness, but at this time she was not so thin as she had been a short time before. The catamenia had not appeared since the month of May; but, with the exception of considerable loss of strength, her bodily health was now on the whole tolerably good.

On her recovery from the torpor she appeared to have forgotten nearly all her previous knowledge; everything seemed new to her, and she did not recognise a single individual, not even her nearest relatives. In her behaviour she was restless and inattentive, but very lively and cheerful; she was delighted with everything she saw or heard, and altogether resembled a child more than a grown person.

In a short time she became rather more sedate, and her attention could be longer fixed on one object. Her memory too, so entirely lost as far as regarded previous knowledge, was soon found to be most acute and retentive with respect to everything she saw or heard subsequently to her disorder; and she has by this time recovered many of her former acquirements, some with greater, others with less facility. With regard to these, it is remarkable that though the process followed in regaining many of them apparently consisted in recalling them to mind with the assistance of her neighbours, rather than in studying them anew, yet even now she does not appear to be in the smallest degree conscious of having possessed them before.

At first it was scarcely possible to engage her in conversation; in place of answering a question she repeated it aloud in the same words in which it was put, and even long after she came to answer questions she constantly repeated them once over before giving her reply. At first she had very few words, but she soon acquired a great many, and often strangely misapplied them. She did this, however, for the most part in particular ways; she often, for instance, made one word answer

for all others, which were in any way allied to it; thus in place of "tea," she would ask for "juice," and this word she long used for liquids. For a long time also in expressing the qualities of objects, she invariably, where it was possible, used the words denoting the very opposite of what she intended. and thus she would say "white" in place of "black," "hot" for "cold," &c. She would often also talk of her arm when she meant her leg, her eye when she meant her tooth, &c. She now generally uses her words with propriety, although she is sometimes apt to change their terminations; or compose new ones of her own.

She has as yet recognised no person, not even her nearest connections; that is to say, she has no recollection of having seen or known them previously to her illness, though she is aware of having seen them since, and calls them either by their right names or by those of her own giving; but she knows them only as new acquaintances, and has no idea in what relation they stand to herself. She has not seen above a dozen people since her illness, and she looks on these as all that she has ever known.

Amongst other acquirements she has recovered that of reading; but it was requisite to begin her with the alphabet, as she at first did not know a single letter. She afterwards learnt to form syllables and small words, and now she reads tolerably well, and has shown herself much interested in several stories previously unknown to her, which she has read since her recovery. The re-acquisition of her reading was eventually facilitated by singing the words of familiar songs, from the printed page, while she played on the piano. In learning to write she began with the most elementary lessons, but made much more rapid progress than a person who had never before been taught. Very soon after the torpor left her, she could sing many of her old songs, and play on the pianoforte with little or no assistance; and she has since continued to practise her music, which now affords her great pleasure and amusement. In singing, she at first generally required to be helped to the first two or three words of a line, and made out the rest apparently from memory. She can play from the music-book several tunes which she had never seen before; and her friends

are inclined to think that she now plays and sings fully as well, if not better, than she did previously to her illness. She learnt backgammon, which she formerly knew, and several games at cards with very little trouble; and she can now knit worsted, and do several other sorts of work; but with regard to all these acquirements, as already mentioned, it is remarkable that she appears not to have the slightest remembrance of having possessed them before, although it is plain that the process of recovery has been greatly aided by previous knowledge which, however, she seems unconscious of having ever acquired. When asked how she had learnt to play the notes of music from a book, she replied that she could not tell, and only wondered why her questioner could not do the same.

She has once or twice had dreams, which she afterwards related to her friends, and she seemed quite aware of the difference betwixt a dream and a reality; indeed, from several casual remarks which she makes of her own accord, it would appear that she possesses many general ideas of a more or less complex nature, which she has had no opportunity of acquiring since her recovery.

In this way she has continued slowly but progressively to improve, and it is now considerably more than two months since she recovered from her sleep. Her bodily health has since then undergone little change; she is still liable to be fatigued by slight exertion, after which she is inclined to sleep; but in this state she never remains long except during the night, when she sleeps like another person. The catamenia have twice appeared, viz. in September and in October, at both times to a greater extent than usual; her bowels still require laxative medicine; but her appetite continues good, and she has evidently gained flesh since her recovery.

*Postscript* (March 1879).—After a time Mrs. H. was able to return to her home, in England, where she passed the rest of her life happily with her husband, and gave birth to a daughter, who survives her. She was in the habit of corresponding by letter with her friends at a distance, and lived on the most agreeable terms with her immediate neighbours, by whom she was held in much regard on account of her kindly nature and charitable work.

OBSERVATIONS ON NEURITIS AND PERI-NEURITIS  
OF SOME OF THE CRANIAL NERVES.

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2. *Auditory Neuritis*.—The series of symptoms generally known as Menière's disease appears to me to be owing to a considerable variety of anatomical lesions, and the term which has been given it, in order to mark the merit of the illustrious French aurist who first drew attention to this class of cases, is therefore somewhat of a misnomer, as it does not designate a pathological entity. It is true that we possess at present only a few and meagre post-mortem records of such cases; yet the clinical symptoms are sufficiently striking to enable us to decide that they cannot all be owing to one and the same morbid process, and that the only connecting link between them is that their seat is in the membranous labyrinth. I think it would be best to confine the term, "Menière's disease," to apoplexy of, or more properly speaking hæmorrhage into, the labyrinth, as most of the cases described by Menière belong to that class, and the only case in which he obtained an autopsy was also one of them. This was the case of a young woman who took cold at the period of menstruation, while riding on the top of an omnibus, and was suddenly seized with deafness, persistent vertigo, and vomiting. She died on the fifth day, and it was found that the centres of the nervous system were perfectly healthy, but that the semicircular canals on both sides were filled with a clot of blood, replacing the endolymph. This clot extended in a minor degree into the vestibule, but



did not spread as far as the cochlea. Similar cases, but in which the cause was injury to the bone, causing hæmorrhage and subsequent inflammation, have been recorded by Politzer and Voltolini. In one of them the injury was a blow on the temple, in the other a fall on the back of the head. In other cases the symptoms have come on without any apparent cause, and it is not improbable that in these the hæmorrhage may have been owing to rupture of miliary aneurisms of the labyrinthine arterioles, and therefore be analogous to the ordinary form of cerebral hæmorrhage. Such hæmorrhage, however produced, causes an increase of the intra-auricular pressure, which is quite sufficient to account for such symptoms as deafness, tinnitus, unsteady posture, so that the patients are unable to stand or walk firmly, and fall down, or are only prevented from falling by the bystanders; together with fainting, nausea, vomiting, and in some cases insensibility.

There are, however, unquestionably other pathological processes giving rise to symptoms similar to those just described. One of them I believe to be simple hyperæmia of the labyrinthine blood-vessels, which I suspect in cases where the symptoms are sudden in their onset, not very severe, and transitory in their appearance. Sometimes the attack is entirely over in a quarter of an hour, without leaving any traces; and in such cases it is impossible to assume the existence of hæmorrhage. Another and more important lesion I believe to be inflammation, which may be idiopathic, *i.e.* occurring without any apparent cause, or symptomatic, *i.e.* coming on in the course of severe systemic affections, more particularly pyæmia, the eruptive fevers, erysipelas, typhoid and puerperal fever, constitutional syphilis, &c. Again, inflammation may spread contiguously to the labyrinth from other parts, such as the middle ear, when this is affected by acute catarrh and purulent inflammation; and from the membranes of the brain, in sporadic or epidemic cerebro-spinal meningitis. It is also worthy of notice that somewhat sudden deafness occurs in connection with infantile eclampsia, and it appears highly probable that in such cases the real cause of the convulsive seizures, as well as of the deafness, is irritation or inflammation of the labyrinth, and that the fits are caused by the extreme liability of infants

to convulsive attacks from whatever cause, whether central or eccentric. Inflammation of the labyrinthine expansion of the auditory nerve I believe to be more common than is generally thought, and it has no doubt often been confounded with congestion or inflammation of the brain, and even been put down as an attack of severe dyspepsia and congestion of the liver. The chief difference in the clinical symptoms of hæmorrhage and inflammation is that the symptoms are not so severe and sudden in their onset in the latter, that they continue for a more considerable time, and become gradually more developed.

I will now relate the details of a case of what I believe to have been neuritis of the labyrinthine expansion of the auditory nerve, which occurred in a patient who was admitted into the hospital under my care in January 1876, suffering from progressive locomotor ataxy.

H. W., aged 32, married, father of three children, and a butcher by trade, had been in good health until the commencement of 1875, when he began to feel poorly, and suffered from a troublesome form of indigestion, with nausea and loss of appetite. In May of the same year he suddenly began to squint and see things double, evidently from paralysis of one of the ocular muscles, although it would be impossible to determine at present which one of them was affected. These latter symptoms lasted only for a few days and then left him; but shortly afterwards he was affected with vertigo and a roaring noise in the head. There was also a feeling of sickness, but no vomiting. Within a few days the tinnitus increased considerably, and appeared to him like thunder, or as if there were explosions of gunpowder in his head; at other times it resembled the ringing of bells and screaming of whistles. There was at no time any loss of consciousness. This severe form of tinnitus lasted for rather more than a month, during which time the hearing of the patient, which before then had been perfectly good, was *gradually* much diminished, and at the end of the period mentioned he found himself completely deaf. By this time the vertigo had left him, but, on going about, he noticed that he did not walk as well as before, more particularly in the dark, and was apt to stumble, especially on turning round; and he felt the peculiar sensation as if walking on

cotton or bladders. Pain of a character peculiar to ataxy began to shoot through the lower extremities, more especially in the night, and on exposure to wet or cold. The ataxy increased rapidly, in spite of medical treatment; so that he became completely helpless; and when he entered the hospital, in 1876, he had already reached the third stage of the disease, in which not only the co-ordination of movements but also muscular power suffers.

The examination of the patient proved unusually troublesome, as he was stone-deaf, and all questions had therefore to be written down for him on a slate. He was found to be utterly insensible to the shrillest and loudest noise, such as that of a cab-whistle blown just behind him, as well as to the sounds of musical instruments. He could not hear a watch tick when it was applied to the external ears or the cranial bones around; nor did he perceive the sound of a tuning-fork applied to the vertex and to the teeth. On applying the constant voltaic current to the ears, however, a distinct sound was perceived on making with the cathode and breaking with the anode. This sound the patient likened to a "blowing" or "ringing" noise, and it appeared to continue for some seconds after the current had commenced and ceased to act. This was over and above the habitual tinnitus, which never left the patient, and which was now of a moderate kind, resembling the flowing of water. The patient spoke intelligibly, and although he could not hear himself speak, he had no deficient or altered intonation of the voice.

The physiognomical expression was peculiar. His features appeared in perfect repose, and unimpressible, except when a question in writing was put to him. Having noticed a similarly statuesque expression in anæsthesia of the fifth nerve, from loss of cutaneous and muscular sensibility, I carefully tested the sensibility all over the face, but found it perfectly normal; and the total absence of physiognomical expression was therefore in this case owing to the patient being as it were shut out from the world, and his being indifferent to what went on around him.

I will here remark at once that some time later I had the advantage of Mr. Dalby's opinion on the state of the patient's

ears. He confirmed my diagnosis of the nervous origin of the deafness, as he found the external and middle ear, including the Eustachian tube and the tympanum, perfectly healthy. The conduction of sound was good, but the perception of it faulty, and he therefore thought the deafness due to change in the nervous structures, either in the labyrinth or intracranial.

The patient had not inherited any tendency to nervous affections such as paralysis, insanity, or neuralgia. He had always been a steady, hard-working man, not given to alcoholic or venereal excesses. He had never had syphilis or gonorrhea. He never smoked. He had, however, in his trade, as a butcher, been obliged to go about a great deal in all kinds of weather, and in the small hours of the morning, and had lately had much anxiety about money matters.

There were no symptoms indicating cerebral disease; the intellect, memory, and speech being quite normal, and all the cerebral nerves, with the only exception of the auditory, were in full functional activity.

The spine was not tender to pressure or percussion, nor was there any spontaneous pain in it; and the pain in the limbs was less marked than it had been some time ago. There was incomplete cutaneous anæsthesia from the waist downwards to the feet, and also incomplete muscular anæsthesia. Tickling the soles produced no reflex movements, and pinching the gastrocnemius and rectus femoris produced hardly any sensation. The muscles were flabby and somewhat wasted, but responded freely to the voltaic and faradic currents. The patient could not walk at all, except when supported by two persons, and even then he had the greatest difficulty in stepping out, the peculiar jerking gait of ataxy being perfectly discernible. The helplessness was so great that it verged on paralysis. He could only stand when supported by two sticks, and when he closed his eyes he reeled like a drunken man. Yet he could, when lying down or sitting on a chair, move his legs and feet more freely than a patient can do who is suffering from myelitis or a high degree of spinal congestion.

The sexual power had been gradually lost during the last six months, and the bladder and rectum likewise participated

in the disease. There was great difficulty in passing water, the patient having to strain for fifteen or twenty minutes before he succeeded in voiding a few ounces of urine. Occasionally there was incontinence. The urine was habitually neutral, and contained a large excess of urea and phosphates, but no albumen or sugar. The bowels were confined, and when purgatives were administered they often acted so rapidly that the fæces were voided before the patient had time to reach the commode.

The upper extremities were unaffected, with the exception of a slight feeling of numbness in the third and little finger of the left hand. The heart and lungs were healthy. The appetite, however, was very bad, and digestion much impaired; the tongue was furred. There was tenderness in the right hypochondrium, and increased dulness in the region of the liver. The patient was considerably emaciated, and had a sallow and dyspeptic complexion. He was ordered nitrate of silver, hypophosphite of soda, and podophyllin. For two months no change for the better took place; on the contrary, the helplessness increased, and there was more frequently than before incontinence of the bladder and bowels.

Towards the end of March the patient was suddenly taken with vertigo, followed by coma. There were no convulsions. The complexion became of a deeper sallow, and after a time dark brown; the tongue had a dirty brown coat; there was involuntary discharge of the excretions. The pupils were very small. The right hypochondrium was very tender, the pulse 120, the temperature  $99.5^{\circ}$ . In three hours the coma was less severe, and he was given five grains of Plummer's pill, followed by five grains of carbonate of ammonia every four hours in mixture. On the next day the coma was not profound; the morning temperature was  $100.2^{\circ}$ , pulse 110; the evening temperature  $101.5^{\circ}$ , pulse 120. The next day the insensibility had again lessened; the patient could be roused by talking loud to him; the morning temperature was  $99.6^{\circ}$ , the evening  $101.4^{\circ}$ . On the day after that the improvement was more decided; the expression brighter, the complexion less dark, consciousness had returned, the evening temperature was only  $99.2^{\circ}$ . On the fifth day the patient ate with a good

appetite, and retained the fæces and urine for the first time. The temperature was  $99.2^{\circ}$ , the pulse 90. On sitting up he felt giddy.

From this time forward he rapidly improved, and a week afterwards he was again in the same condition as before this attack. The medicines he had previously taken were now discontinued, and the liquid extract of ergot prescribed in half-drachm doses three times a day. This had an excellent therapeutic influence, since under its use a steady improvement took place in all the spinal symptoms. The remedy was gradually pushed up to a drachm three times daily, and was only discontinued once for a few weeks, after having been taken for three months, as the patient then complained of general malaise, with loss of appetite, and had a feeble pulse. The citrate of iron and quinine was then substituted for the ergot, with good results as far as those symptoms were concerned; but there was now no further improvement in the spinal disease, and the ergot was therefore resumed a few weeks afterwards in forty-minim doses. It was altogether taken for eight months; and at the end of that time the patient had entirely recovered from all symptoms of ataxy; only the deafness and the tinnitus continued exactly the same. He was discharged in February 1877, being then able to walk easily three or four miles; and when seen twelve months later, had been able to attend to his business just as before the commencement of the affection, and had had no further attacks of vertigo or insensibility, or, in fact, any other ailment.

*Remarks.*—It is now well understood that the auditory nerve is not simply a nerve of special sense, but has a distinct relation to certain centres which regulate the equilibrium of the head and the body. These functions appear to be so divided that the cochlear branch of the portio mollis is the real nerve of hearing, while the vestibular branch of the same regulates equilibration. Section of the auditory nerve in the skull, before it has divided itself into its two branches, causes both deafness and loss of equilibrium. A frog whose auditory nerves have been cut, may still jump when irritated, but will not regain its proper position afterwards; it falls on its

back or side, and rolls over and over, without being able to steady itself. The same is seen when the semicircular canals in the membranous labyrinth are injured, while injury of the same in the osseous labyrinth produces no such effects. Pigeons in whom Flourens destroyed these canals, heard as well as before, but showed loss of equilibration: while they lost their hearing, and showed no loss of equilibrium, when the cochlea was injured. In opposition to this, Menière taught that disease of the semicircular canals alone would cause deafness as well as vertigo; but it appears now well-established by the researches of Goltz, Vulpian, Crum Brown and others, that these canals have nothing to do with hearing, but serve to maintain the normal attitude of the body. These exceedingly difficult and intricate experiments are somewhat facilitated by adopting Vulpian's plan, which consists of previously feeding the animal with madder, whereby the canals are stained bright red, and are therefore rendered distinct from the bones in which they are enclosed, and which have a much paler colour. Each of the three canals appears, according to the most recent observers, to have a different function. Thus, division of the superior canals causes the head to be rapidly moved forwards and backwards, and there is tendency to execute a somersault forwards, heels over head; similar phenomena being induced by lesion of the anterior portion of the middle lobe of the cerebellum. Lesion of the external or horizontal canals produces rapid oscillations of the head and eyes from one side to another, and tendency to spin round; phenomena corresponding to those after injury to the lateral lobes of the cerebellum. Finally, destruction of the posterior or inferior canals leads to rapid movements of the head backwards and forwards, with tendency to execute a somersault backwards, head over heels; and lesion of the posterior portion of the middle lobe of the cerebellum will cause corresponding signs. That there should be such correspondence is readily accounted for by the microscopical investigations of Lockhart Clarke and Meynert, who have shown that the auditory nerve is, through the restiform bodies, in direct communication with the cerebellum, which we look upon as the central organ of equilibration.

Integrity of the labyrinth is therefore shown to be necessary for the maintenance of equilibrium of the head and the body; and labyrinthine impressions are shown to be more important towards securing that end than any visual or tactile impressions, which are also concerned in it. These impressions appear to be dependent upon variations of pressure in the endolymph which fills the membranous canals. Pressure or tension of this liquid excites the terminations of the nerves in the canals and their dilatations or ampullæ. In accordance with the law of gravity, the endolymph distends mostly those portions which lie deepest; and the pressure naturally varies with the movements and position of the head, so that each position corresponds to a definite state of nervous stimulation. The perception and consciousness of this state by the brain constitutes the sense of equilibrium, and thus serves to regulate the movements of the body. As soon, therefore, as any injury to the canals has taken place, the brain ceases to receive accurate information of the position of the head, and is rendered unable to calculate and direct its movements in a proper manner. This disturbance is temporary when lesion of the canals exists on one side only, but is rendered permanent when both sides are affected. Loss of one eye does not produce blindness; and the organ of one side may, by a certain amount of training, become efficient to inform the brain of the position of the head and the body.

Dr. Ferrier<sup>1</sup> finds the auditory centre to be situated in the superior temporo-sphenoidal convolution of the hemispheres. When this part is faradised in the monkey, there is a sudden pricking up of the opposite ear, wide opening of the eyes, dilatation of the pupils, and turning the head and eyes to the opposite side—signs resembling the sudden start and look of surprise that are caused when a loud sound is made suddenly close to the animal's ears; both being indications of subjective auditory sensation. Faradisation of the corresponding part of the brain in other animals whose habits are such as to make their safety depend upon the acuteness of their hearing, as for instance the rabbit and the wild jackal, causes even more striking effects, viz. in addition to those already mentioned, a quick

<sup>1</sup> Loc. cit. p. 171.



start or bound as if to escape from danger which might be indicated by loud or unusual sounds. Destruction of this convolution on both sides causes complete deafness; unilateral destruction of it renders the animal deaf on the side opposite the lesion.

Taking into account these several facts, we cannot experience any difficulty in arriving at a definite conclusion concerning the precise seat of the pathological lesion in the case which I have just related. It was evidently not located in the superior temporo-sphenoidal convolution, which, although the centre of hearing, has nothing to do with the equilibration of the body; nor could we consider the case one of cerebellar disease, the cerebellum being only the central organ of equilibration, but having nothing to do with the sense of hearing. The lesion must therefore have been seated in the auditory nerve, which presides over both hearing and equilibration, and destructive lesions of which will cause deafness as well as vertigo.

At what part of the anatomical distribution of the *portio mollis* was the disease located? Evidently not at its root in the medulla, because there it is contiguous with the sentient root of the fifth nerve, and there would therefore no doubt have been anæsthesia of the face together with the deafness. A case of this latter kind has been described by Professor Moos, in the '*American Archives for Ophthalmology and Otology*.'<sup>1</sup> Nor was it likely that the nerve-trunk was affected where it emerges from the lower border of the *pons Varolii*. I believe this portion of the nerve to have been healthy, because there was galvanic response on applying the voltaic current to the ears; and such response appears to be absent in destruction of the nerve-trunk. We are therefore led to the conclusion that the disease affected the labyrinthine expansion of the nerve, comprising its branch for the cochlea as well as for the vestibule.

The last point we have to consider is the nature of the pathological lesion in the labyrinth. This I believe to have been an acute inflammation, as there was a period extending over rather more than a month in which there were evident

<sup>1</sup> Vol. ii. p. 199.

signs of special hyperæsthesia of the labyrinthine expansion of the nerve, which were followed by special and permanent anæsthesia. These symptoms correspond very closely to those which I have observed in acute olfactory and trifacial neuritis. The bad form of indigestion with nausea, loss of appetite and giddiness, with which the illness commenced in the first instance, was probably due to hyperæmia of the auditory nerve. This increased suddenly, perhaps, in consequence of some particularly severe exposure to the inclemencies of the weather, which the patient's occupation necessitated, to inflammation, which lasted the usual term of acute neuritis, viz. from four to five weeks. The *gradual* loss of hearing during the period just mentioned speaks against hæmorrhage in the labyrinth, in which deafness is more suddenly developed. The cord-affection which followed the attack of auditory neuritis was likewise of a more markedly inflammatory character than is usual in ataxy, as the symptoms became developed with far greater rapidity than is seen in the large majority of cases. The intercurrent attack of illness which the patient had at the end of March, while in the hospital, was, in my opinion, owing to an attack of hæmorrhage into the labyrinth. The attack commenced with sudden loss of consciousness, accompanied with some rise in the temperature, and left no further traces. The hearing having already been completely destroyed, no further damage could be done in that quarter.

3. *Peri-neuritis of the Portio Dura*.—One of the commonest affections of the cranial nerves is paralysis of the portio dura, more especially that form of it which is caused by the sudden influence of cold, and the more prominent symptoms of which are so well known that I need not describe them here. There are, however, several points connected with this affection on which still much obscurity prevails, and to which I will briefly refer, in the hope of contributing somewhat to their elucidation. I will, however, confine myself in this place to a consideration of that form of facial palsy which I consider to be owing to rheumatic peri-neuritis of the nerve, without going into other forms of the complaint which are produced by injury, such as wounds, surgical operations about the face,

application of forceps during delivery; or by pressure from neighbouring parts, such as abscess, swollen glands, tumours, periostitis, or paralysis induced by cerebral disease.

Although post-mortem examinations of cases of rheumatic peri-neuritis of the portio dura are as yet entirely wanting, yet there can be no doubt that this palsy is really owing to an inflammatory swelling of the peri-neurium of the nerve, with more or less considerable effusion of lymph. This is shown by the mode of its onset, which is generally sudden, after some unusual exposure to cold, while in some cases it is only established a day or two after the cause has acted. In the commencement there may be pain about the face, headache, twitches in the affected muscles, and tinnitus aurium; but as the portio dura is almost exclusively a motor nerve, there is naturally much less pain than occurs in neuritis of purely sentient or mixed nerves. The principal symptom is therefore the palsy, which is more or less severe and extensive, according to the intensity of the cause which produces it, the condition of the patient at the time he becomes subject to the affection, and more especially according to the anatomical seat of the lesion.

Broadly speaking, we may divide all these cases into two large classes, viz. those where the inflammation occurs outside, and those where it occurs inside, the Fallopian aqueduct. In external peri-neuritis, which affects the nerve immediately after having emerged from the stylomastoid foramen, the pressure of the inflammatory swelling upon the contents of the nerve-tubes is not very great, as the nerve is there surrounded by soft parts, and therefore not so apt to be injured by squeezing; and in consonance with this we find that facial palsy from external neuritis generally gets well in two or three weeks. This would be inconsistent with the assumption of actual neuritis, in which the cylinder axis is destroyed. The most prominent symptoms of this variety of the affection are palsy of all the facial muscles of one side, and loss of reflex excitability in them, on irritating the conjunctiva of the eye and eyelids, or the skin of the face. The electric response of the muscles, both to the faradic and voltaic current is normal, and there is an absence of all symptoms

implicating the senses of taste and hearing, and the soft palate.

That the seat of this external peri-neuritis is not in the peripheral branches of the nerve, or the pes anserinus, but in the trunk of the nerve, shortly after it has left the skull, is plainly shown not only by the paralysis affecting all the muscles of the face, but also by there being no affection of sensibility. If the seat of the inflammation were more peripheral, some facial muscles would no doubt be spared, and sensation in the face would suffer, from the intimate connection which exists between the branches of the facial and of the fifth nerve. A final proof of what I have just advanced is that, in some few patients of this class, paralysis of the small muscles which move the ear has been observed. These muscles are supplied by the posterior auricular nerve, which is the first branch given off by the portio dura after its exit from the stylomastoid foramen, and which also gives a twig to the posterior belly of the occipito-frontalis muscle. Most people are indeed unable to move their ears, but some few can, and in some of them inability to move one ear has been observed under these circumstances.

The second large class of cases of facial palsy is that which is owing to *internal peri-neuritis* affecting the nerve at some point during its transit through the Fallopiian canal. In such cases the cause and degree of the inflammation may be exactly the same as in external peri-neuritis; yet the results are more serious, for the nerve is there no longer surrounded by soft parts, but enclosed in rigid walls. The same amount of swelling will therefore give rise to a much more severe compression of the contents of the nerve-tubes, and thus cause more degeneration and wasting in the same. The prognosis of internal peri-neuritis is therefore much less favourable than that of the external variety of the disease; and it is an interesting fact that, by a careful electric exploration of the affected nerve and muscles, we are enabled, within the first week or two, to determine with a great degree of accuracy whether the patient is likely to recover quickly, or only within from three to six months. In internal peri-neuritis, where there is more squeezing, we discover generally within eight or

ten days from the commencement of the affection, and sometimes even sooner, that peculiar alteration in the faradic and voltaic excitability of the nerve and muscles which I have called the *wasting-test*, and the importance of which cannot be overrated.

The distinctive feature of this test is that the induced or faradic current loses all influence upon the paralysed nerve and muscles, so that, on comparing the two sides of the face in this particular, faradisation of the portio dura and all the individual muscles of the face will cause these to move on the healthy side, but remains ineffectual on the paralysed side. Again, the constant voltaic current loses in a similar manner its influence upon the *nerve*, but acquires increased action over the individual muscles which are paralysed. It is therefore seen that a constant current, which is too feeble to produce an effect on the healthy muscles, will cause the paralysed ones to act very readily. This fact was first pointed out by Baierlacher. At the same time the character of the voltaic contraction is altered. It does not occur with that lightning-like rapidity which it assumes in the healthy muscle, but is sluggish, tardy, inert, and recalls to one's mind those muscular contractions which are seen in physiological laboratories in frogs whose nerves have been paralysed by woorare or coniine.

These phenomena are only present where there is muscular wasting owing to nerve-lesions, so that the term *wasting-test* appears to be an appropriate one to designate them. The *absence of the wasting-test is characteristic for external, and its presence for internal peri-neuritis*, the reason for this being that in external inflammation there is less pressure on the nerve, and consequently no muscular atrophy; while in internal inflammation the nerve is thoroughly squeezed, and thereby loses its nutritive or trophic influence over the muscles supplied by it. In the former case, therefore, the patient generally gets well within two or three weeks, while in the latter recovery will be protracted over from three to six months and even more; or the nerve may not recover at all, but the paralysis may in its later stages become complicated with muscular rigidity, twitches and atrophy, which imparts a most peculiar expression to the countenance. Such consequences are not

simply owing to disuse of the muscles, for they do not occur in facial palsy from cerebral origin even when this has lasted much longer, but are due to the withdrawal of nutritive power, from nerve lesion.

Internal peri-neuritis of the portio dura may be subdivided into several forms, according to the exact spot in the Fallopian aqueduct where it occurs; and in no other disease can we trace the anatomical distribution of the inflammation, simply from the clinical symptoms, in a more minute manner than in the one to which I am now drawing attention. The Fallopian aqueduct may for this purpose be divided into three different sections, the first of which reaches from the stylomastoid foramen to a point just before the origin of the chorda tympani; the second embraces the chorda and the Stapedian nerve; and the third corresponds to the ganglion geniculum, where the petrosal nerves take their rise. The nerve may, however, also be subject to inflammation at the base of the brain.

(a) Where the inflammation takes place in the *first section* of the Fallopian canal, below the origin of the chorda, we have the same symptoms as in external peri-neuritis, viz. facial palsy and loss of reflex excitability; but in addition to this the phenomena of the wasting-test in the paralysed muscles. Erb has drawn attention to certain hybrid forms of the disease in which the wasting-test is somewhat blurred and indistinct, there being no actual loss, but only diminution of faradic excitability of the nerve and muscles, which latter show increased voltaic response. This indicates that the pressure on the nerve is not very great, and that tolerably rapid recovery may be expected. Such cases probably correspond anatomically either to a severe form of external, or to a mild form of internal peri-neuritis in the first section of the Fallopian canal.

(b) If the *second intra-Fallopian section* of the nerve be inflamed, we find, besides the foregoing symptoms, a peculiar modification of the sense of taste, which has to be ascribed to the chorda tympani being involved in the inflammation. The patient then experiences an acid, or metallic, or bitter, or simply a disagreeable taste, in the corresponding half of the tongue; and on testing the anterior portion of that organ

with saline, bitter, sweet, and acid substances, it is found that they are not perceived there. The back part of the tongue which is supplied by the glosso-pharyngeal nerve, and the opposite side of the organ, the nerves of which are unaffected, continue as keenly sensitive to sapid substances as previously, so that the patient before the examination is often not aware that the sense of taste is impaired. The chorda tympani proceeds from the portio dura to the lingual branch of the fifth nerve, and ends in the submaxillary ganglion. It is no doubt owing to this latter circumstance that, coincidently with the loss of taste, there may be great dryness in the corresponding side of the mouth, owing to arrested secretion of saliva in the submaxillary gland. The experiments of Nawrocky have rendered it probable that the chorda also influences the secretion of saliva in the sublingual gland, and that the parotid receives secretory fibres from the superficial minor petrosal nerve, so that the dryness of the mouth is amply accounted for. Eulenburg<sup>1</sup> has suggested that the dryness of the mouth may be owing to its being always a little open, in consequence of the paralysis of the orbicularis oris, whereby the evaporation of saliva is accelerated; but this theory seems to me unsatisfactory, as dryness of the mouth does not occur in facial palsy from cerebral or medullary origin, where, nevertheless, the mouth is somewhat open; and I hold therefore to the view that the symptom is owing to paralysis of the chorda tympani.

Another interesting symptom of neuritis in the second intra-Fallopian section of the portio dura is *hyper-acusis*, or oxyokoia, that is, hyperæsthesia of the sense of hearing, which has to be attributed to paralysis of the Stapedian nerve. This nervelet, which leaves the portio dura a little above the chorda tympani, supplies the Stapedius, or levator tympani muscle. The pressure in the tympanum and the tension of the drum of the ear are regulated by two different muscles which receive their nervous supply from different sources, viz. the levator, which is animated by the Stapedian from the portio dura, and the tensor tympani, which receives a branchlet from the otic ganglion of the fifth nerve. This latter muscle does therefore

<sup>1</sup> 'Lehrbuch der Nervenkrankheiten.' 2nd Edition. Berlin, 1878. Vol. ii. p. 115.

not participate in the paralysis, but attains increased action by removal of the influence of its antagonist. The drum of the ear therefore acquires a higher degree of tension, the pressure in the tympanum is intensified, and excessive keenness in the appreciation of all musical sounds is the result. These sounds also appear somewhat lower than they really are, besides which there is a considerable degree of tinnitus, and an uncomfortable sensation of pressure in the internal ear. Whenever therefore we meet with hyperacusis in affection of the portio dura, we are justified in assuming that the seat of the disease is above the origin of the Stapedian nerve. This peculiar affection of hearing is entirely independent of any disease of the auditory nerve itself, and of internal purulent otitis, which are such frequent complications of facial palsy.

(c) Where the peri-neuritis affects the *third* intra-Fallopian section of the portio dura, at its bend, and implicates the ganglion geniculum, near the internal end of the aqueduct, another important symptom makes its appearance. At this point the portio dura sends off the large superficial petrosal or Vidian nerve, which proceeds to the sphenopalatine ganglion, and through it to the soft palate, where it supplies the levator palati muscle. Vidian paralysis therefore causes deficient action of the velum palati, which droops on the paralysed side, and does not respond well to reflex or electrical excitation. In some cases it has been found deviated to the other side; and there may be difficulty of deglutition, a nasal twang in the voice, and regurgitation of liquids through the corresponding nostril. The uvula may also be found deflected either to the healthy or to the paralysed side. We can easily understand why it should be pulled to the healthy side, from preponderance of the healthy muscle; but why should it be found deflected to the paralysed side? Dr. Sanders has suggested that this is owing to increased action of the pharyngo-palatinus muscle, whose fibres terminate at the base of the uvula, and which is the antagonist of the levator palati. This muscle would therefore act more powerfully on the paralysed than on the healthy side, receiving its nervous supply from a different source. The matter is, however, rendered very complicated by the circumstance that the uvula is not by any means straight in many



healthy persons; and conclusions from an oblique position of it must therefore be drawn with considerable caution.

Nearly at the same level with the large superficial petrosal, the portio dura sends off the small superficial petrosal to the otic ganglion, and the external superficial petrosal to the sympathetic plexus of the middle meningeal artery; but symptoms owing to paralysis of these nerves have not yet been observed. Peri-neuritis in this third section of the Fallopian aqueduct is rare, and we therefore find that the symptoms just mentioned are absent in the majority of cases.

A common complication of facial palsy is deafness, which is owing to simultaneous affection of the portio mollis. Both nerves may be affected at the base of the brain, or there may be purulent internal otitis, in which case there is generally perforation of the membrana tympani, and purulent discharge from the external ear. If the portio dura becomes inflamed at the base of the brain, where it emerges from the posterior border of the side of the pons Varolii and the lateral tract of the medulla oblongata, there are almost invariably symptoms on the part of other cerebral nerves, more especially the portio mollis. This is seen in syphilitic periostitis, exostoses, and gummatous tumours, and is generally more owing to squeezing from without than to neuritis or peri-neuritis. Facial palsy is also observed in affections of the nucleus of the nerve in the medulla, pons, the cerebral peduncles and hemispheres, but is then generally owing not to inflammation, but to softening, hæmorrhage, tumours, &c., and does not therefore fall into the sphere of the present paper.

The prognosis of peri-neuritis is generally much more favourable than that of neuritis, because in the former, although there is pressure on the nerve-tubes, yet the cylinder axis generally escapes destruction, while in the latter the whole of the contents of the nerve, including its central core, is destroyed. Thus we find that almost all cases of facial palsy ultimately recover, while olfactory and auditory neuritis is rarely influenced by any treatment. It is true that these latter cases are generally only specially treated after the inflammation has subsided, and when the nerve-tubes are left in a state of hopeless decay.

If a case of acute neuritis is recognised in the beginning, it should be treated according to general principles, i.e. by leeches, blisters, and the application of ice as near as possible to the seat of the disease. This should be combined with the internal administration of calomel and opium, in doses of one grain each, several times a day. After the acute stage has subsided, a stimulating treatment must be resorted to, more particularly the application of the constant voltaic current to the suffering nerve. Iodide of potassium may also be given, although there is not much evidence to show that it really is useful in such cases. For peri-neuritis the same rules hold good as for neuritis, and are fortunately more effective in practice.

## AUDITORY VERTIGO.

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### CASE I.—*Ocular Movements during a Paroxysm of Auditory Vertigo.*

THE following case is of interest with regard to the valuable experiments of Cyon. A critical account of these experiments has been given by Ferrier in the second number of 'BRAIN.' There is a paper on Cyon's observations by Croom Robertson in 'Mind,' October, 1878; and one by Crum Brown, 'Nature,' October 10th and 17th.

It will be best to refer first to a case already recorded by Schwabach ('Zeitschr. f. prakt. Med.' No. 11, 1878). I quote the following from Clarence Blake's Otological Review in 'Archives of Ophthalmology and Otology,' vol. vii., No. I., page 113.

"Schwabach reports a case of otitis media purulenta, accompanied by profuse discharge, with swelling and tenderness over the mastoid, severe vertigo upon pressure upon the ear, and in addition the following curious symptom.

"Upon pressing above and behind the auricle, the flow of pus was increased from the external auditory canal without increase of pain, but with marked oscillatory movements of both eyes, the eyes being turned towards the affected side, and slightly downward, the size of the pupils remaining unchanged.

"These movements ceased so soon as the pressure was removed, but were repeated in a lesser degree on syringing the ear. These movements were accompanied by vertigo, as

above mentioned. With the improvement in the disease of the ear, these peculiar symptoms gradually diminished. The author bases his explanation of the contraction of the muscles of the globe upon the experiments of Cyon upon rabbits, and assumes an irritation of the semicircular canals, in consequence of the pressure exercised upon the ear. The transmission of the irritation to the cerebral motor centres corresponding to the muscles is unexplained. The frequency of the oscillations was from 20 to 150 in the minute; their duration depended upon the pressure, but seldom exceeded half an hour."

The following is a note of my patient's case :—

Nov. 16, 1877.—A man, 25 years of age, had his first attack of vertigo four years ago. He was taken when apparently quite well (he was playing cricket) with giddiness; he did not become insensible; he vomited; he had a noise in his left ear. He has since been deaf of that side, but whether he was so before his first vertiginous attack or not he cannot be sure; he has had a continuous "humming" or "buzzing" (he used both words) noise in that ear ever since. During the last four months he has had many attacks.

At his first visit I had only his account of the paroxysm to go by. He said his attacks began by the left eye "being full of specks;" they were not bright nor coloured. Things (i. e. things in the room where he might be) began to pass to the right—he said "from his left eye over his nose." The same things kept passing continually in this direction; *reappearing* on the left, and again and again passing to the right: he did not see them *coming back*. He said that *next* he became giddy and vomited. There was one attack in which he declared that he lost consciousness; but apparently he really did not, as he said he knew people about him; he could not speak. This attack began suddenly: he was giddy, and vomited.

About a fortnight later I saw him again. He had had but one slight attack—"a hot sweat." Whilst telling me that he had been better, he became suddenly ill. I led him to the sofa, as he seemed apparently unable to stand. He lay half-reclining on the sofa. He looked very ill, and said he felt

giddy. I looked at his eyes about two minutes after he began to complain, or rather, I saw them moving, it not having occurred to me to look at them. What I saw is, I think, exactly confirmatory of what he told me on the previous visit, and of what he said after the seizure I witnessed. I asked no leading questions at any time. The pupils were very large, and the two eyes were in movement. Each eye was partially and very slightly rotated to the right in frequent jerks from left to right. The end of a mantelpiece was opposite him. I was much interested in hearing him *volunteer* the remark about the mantelpiece, that "it is going in this way" (he jerking his hand from left to right to show what he meant). After the brief time taken in writing down the above observations—I wrote them down at once—I found that the eyes were still: the mantelpiece had, he said, ceased "going" to the right. The attack was not a severe one. I guess its duration at ten minutes; his pulse was not irregular. I did not however feel it throughout. Towards the end of the paroxysm he perspired; his temperature in the axillas was normal. There was no sickness.

The observation was hurried. I had to attend to the patient as well as to observe his symptoms, but it is worth recording, if only to draw attention again to the desirability of making observations of the eyes during paroxysms of auditory vertigo.

Things I should try to observe, had I another chance, would be more carefully the nature of the jerks—the number in so many seconds. I should ask the patient to tell me how things outside moved, being very careful not to let him know how I expected them to move. Then, after the jerking movements had ceased, I should note whether there were any "negative after-movements of external objects," the analogue of "negative after-images." I made no inquiries in this case. It is possible that the mantelpiece might seem to be slightly moving in the opposite direction when the spasm was over. I ought to have tried to ascertain if my patient's eyes could move as far and as rapidly to the right as they could to the left. But in a bad case of auditory vertigo we should not, I fear, be able, on account of the great pros-

tration and misery of the patient, to make any precise observations.

The patient was much benefited, for a time at least, when the treatment advised for such cases by Charcot was begun—large doses of quinine.

On January 3rd, 1878, I prescribed four grains of quinine three times a day. On his next visit he said he had not been so well for four months. I saw him some months later; he said the quinine restored him wonderfully, and he took it when “a little queer.” This queerness was “going a little giddy;” “things seeming to go” to the right. Moreover, he had had three stronger attacks.

I saw him again February 11th, 1879. He still has slight attacks. He now and then takes the quinine, which relieves him.

He now says that after a giddy attack he passes a large quantity of urine of a lightish colour.

On February 26th, 1879, Mr. Laidlaw Purves was so kind as to examine the patient's ears; the following is Mr. Purves' summary of an elaborate examination:—

“The membrana tympani is congested, but not more so than may be accounted for by the catarrh from which the patient is at present suffering, or by the constant concussions to which he is subjected during the exercise of his trade. Apart from the catarrh, congestion, and dulness of the sensation of the acoustic, the organ is healthy.”

It is at first glance difficult to understand how it is that persistent changes in the ear, or changes which vary little, can be the cause of *paroxysms*. Sydney Ringer suggests that one factor in the development of the paroxysm of migraine is a diminished resistance of nervous centres. It may be, similarly, that the aural disease varies little, but that the attack is determined by diminished resistance of nervous centres during general enfeeblement of health. There are chronic cases of auditory vertigo, the patient being often a very little giddy, and scarcely perceptibly unsteady in his gait; in these certainly the vertigo varies with the general health, being worse when the patient is below par, and sometimes passing off altogether when he is invigorated by a well-spent holiday. The arti-

ficial invigoration by quinine may cure, or cure temporarily, by enabling the centres to resist the "irritations" starting from the ear. The occurrence of noises in the ear, after excessive doses of quinine, may perhaps be owing to exhaustion consequent on over-stimulation, and thus not be a fact discrepant with the opinion that large doses of quinine increase the resistance of nervous centres.

By a very superficial process of reasoning we might suppose that salicylate of soda in small doses might be a useful remedy in auditory vertigo; for in the doses given for rheumatic fever it causes great noise in the ear. We shall see that in the next case an illness during which salicylate was taken was followed by constant noise in a faulty ear; but the circumstances of this case are too complex for any exact conclusion to be drawn as to the action on the ear of the drug mentioned.

Several of my medical friends tell me, referring to my account of the paroxysm I witnessed, that the mantelpiece "ought to have appeared to move to the left—in an opposite direction to that of the eyes." That the eyes moved to the right I am sure; and the patient always told me that things passed to the right. I never asked him any leading questions. Until these objections were made, it never occurred to me that there could be anything incongruous betwixt his report and the direction of movements of his eye, and now I do not see anything discrepant.

CASE II.—*Noise in the Right Ear, with tendency to walk to the Left side, consequent on discharge of a heavily loaded gun near the Right Ear.*

My friend, Mr. Lewis Mackenzie, knowing my interest in Menière's disease, obtained for me details of a case which seems to me like an experiment bearing upon the production of certain symptoms of that disease. Mr. Mackenzie noticed when walking with, and to the left of a medical man (whom we shall henceforth call the patient), that he was gradually pushed to the left by the patient; on another occasion, the sides being reversed, he noticed that he was gradually pulled to the left. On inquiry, he found that the patient, in his gait, always tended to the *left*, and that he was deaf of the *right* ear.

Now for the history of these and other symptoms. The notes were taken by Mr. Mackenzie, February 1876. The case is all the more valuable in that the patient is a medical man, and in that the examiner into it is a methodical observer.

J. W., aged 34, surgeon, in very active practice, and apparently in perfect health, gives the following history :—

In October 1871 he was out shooting, and the birds being very wild, his host asked him to put in his gun some extra-loaded cartridges; this he did, but after firing his first two shots almost together with these heavily-loaded "pin-firing cartridges," he thought for a moment that his gun had burst, so great was the noise to him. For an appreciable interval of time, a moment or so, he felt giddy and "stunned," as it were, but immediately he recovered himself, and, on examining his gun, found it all right, and he walked on. At the discharge of the gun he did not fall, nor had he to grasp anything nor to ask any one to help him to stand; he did not vomit, nor even feel sick. But, as he began to walk on, he noticed that he had a "humming noise," a "frightful row," in his right ear, and that his right leg crossed over his left every time he put it forward, so that he could only with great difficulty walk straight. He finished his shooting, but only killed one more bird, as his aim was nearly always wrong. When he got home, he found that he was almost completely deaf of his right ear, and that there was in it a "buzzing," or a "humming like a bee."

This was a source of constant annoyance to him: he says it was so bad that it seemed, to use his own words, "to affect my memory, and for a time I could not recollect names nor add up any figures well. In the morning when I awoke it used to trouble me most, and then the sound was much higher pitched than after exertion. It has always been much more troublesome after a hard day's work, and especially after any wine-drinking or after smoking more than usual." The patient plays the violin, and has a very correct ear for musical tones; he says the noise has in no way affected the acuteness of his musical hearing. The noise is not altered when eating; and he thinks he has all along heard high-pitched or low-pitched sounds equally well, but has never tested the ears separately.



The giddiness and reeling in his gait towards the left side continued after the accident, more or less severely, for months, and occasionally for years, that is to the present time. It was much worse after any reduction in general health, whether that reduction was from a hard day's work or too much tobacco, &c. : objects did not appear to go round with him, but, he says, "I felt unsteady, obliged to take a step carefully for fear I should go to the left. I was quite conscious of exercising much care to counteract any reel to the left; but when I was at all tired I could not help it, and my wife often had to remind me that I was walking away to the left."

In May 1872, whilst staying at Heidelberg, and after walking fifteen miles in a hot sun, he began to suffer from severe pains in his head, with faintness and constant sickness; he vomited much mucus, &c., violently, and he shivered a good deal for a day or two. This acute head-attack lasted only about forty-eight hours, but left him much reduced in health and very weak for some days. Since that time the patient says he has been gradually improving; his hearing has greatly improved, but it is still impaired (in right ear). He only goes very slightly to the left in walking, and very rarely, and the auditory sensation only annoys him when he is considerably overtaxed.

The patient volunteered the statement that at times his pulse was irregular, and even intermitted as he often laid his finger on his radial, because his heart palpitated very much.

*Feb. 4th, 1876. Examination of the Patient.*—Heart normal, pulse regular, 68, never intermittent during a prolonged examination. Lungs normal; no evidence of abdominal disease; eyes normal; teeth perfect, &c. &c. When asked to stand with his eyes shut and his heels together, his body begins to sway in a circle; eventually *he goes back, and to left side*, to which side he moves his feet; this was extremely slightly marked, but was seen on several consecutive trials. In walking, with one eye shut at a time, no reeling occurred, but the patient expressed his consciousness of having to be careful to prevent going to his left; equally so whichever eye was shut. With both eyes shut he walked without noticeable reel, and well. *Ear.*—Hears watch 2 inches with left ear;  $\frac{3}{4}$  inch with

right ear. Hears tuning-fork when placed on top of head best on side stopped up with the finger, but not so loudly on the right as on the left side. When tested with the violin, he estimates half-tones easily and quarter-tones fairly; and equally well in higher tones as in lower, with each ear.

The patient says that the sound in the right ear is now at times like the "thrill of a low-pitched harp-string after a sharp pull," a sort of twang. At times, it is just as if he had received a sharp box on the ear; at other times there are sounds like the chirping of small birds, in addition to the continuous sounds.

Examined with ear speculum, nothing coarsely abnormal is noticed in either ear. In the right ear, the membrane tympanum is flatter than in the left, not so convex exteriorly; no rupture of either, nor opacity.

I submitted the foregoing account by Mr. Mackenzie to the patient; he was good enough to send me the following. The whole case shows how much disability may result from what is primarily only an ear affection.

*Feb. 1879.*—"Although it is now eight or nine years since the date of my shooting, I have never been really comfortable in my head. The notes you have convey correctly my condition up to about this time last year. My partner had been away six months, and as the spring came on I felt the pressure of work. I had been feeling rather unwell, when one cold day I had to go into the country, and drove myself in my dog-cart; I had slight pain over the left parietal bone, at about midway at its junction with frontal bone. Suddenly everything became a brilliant scarlet colour; this passed off abruptly. The pain increased, and every now and then, when in a room, a greyish mist seemed to pervade the space, so much so that I thought it foggy or smoky. A few days later, I was attacked with a slight feverishness and restlessness at night; had to go out in the morning, but came back almost immediately with a splitting headache (in same place); took to my bed, and suffered extreme pain; temp. 103·5 in the night; had flying pains about limbs; took chloral to relieve pain; slept but little. Next day had fixed pain in both knees, and left eyeball and pain in head considerably better; temp. in morning 101 (4?).

Was too restless to stay in bed ; got up and had return of pain in my head almost immediately, and relief from pain in knees ; was a little delirious at night. Took a few doses of sod. salicyl. Next day better ; collected my thoughts, fancied it was rheumatism, with metastasis to head. Had a profuse sour-smelling sweating. Got rapidly better, and went to work at the end of the week again.

“Now since then I have never known (*unless asleep*) *one moment's* freedom from noise of some kind in my head or ears, or right ear. I can give you this morning a fair idea of my present condition. Last night dined at 7, after a tolerably good day's work ; plain dinner (and less than one glass of ale). After dinner did some reading, smoked one cigar, went to bed at 10.45. Slept tolerably well, as well as usual. In the morning (*as usual*) had that twang (harp-note twang, I call it) in my ear (right) ; at times it seems to come from the occipital region, and is sometimes so loud that, accustomed as I am to it, I have to listen to find out if it is not associated with some other sound. I can't tell you when this twanging sound goes off, it gradually disappears ; but I am *never* without the singing sound (like that which follows a box on the ears). I mean *never*, and it sometimes worries me intensely. I have it occasionally in both, but when that is so, the sound in the left is higher pitched, less loud, not so much of it, and never so lasting. When I have the twang in my head at bedtime, which I often do, I can generally, not always, extinguish it by pressing my head (lying on the side affected) against the pillow ; and I am in the habit of building the ends of my pillow up into a cone, and getting the full weight of my head concentrated in a point at the ear ; directly I turn over the sound is as loud as ever, so I rarely sleep on my left side, for the sound is more than enough to keep me awake. I ought perhaps to tell you the two sounds constantly trouble me at the same time, and then the twanging sound consists of short and regular and frequently repeated sounds, about twenty-eight to the minute, as if a deep-toned harp-string was being repeatedly pulled, and I hear the vibration but not the note. At this moment, 11.30 A.M., my right ear is hissing and singing away merrily enough, and my right

eye feels full and moist, and my head heavy and dull; and were I to attempt any ordinary calculation, I should feel a swimming in my head, and it would require great mental effort to continue the calculation.

“If I stand now with my eyes shut, it is rather difficult to keep myself upright; and although I can’t tell you which side I incline most to, I find myself, or fancy myself, trying to prevent my swaying in a circular direction. Can walk well with both eyes, or either eye, shut. My circulation is good, but easily excited; my bodily health is fairly good, but I am liable to phosphatic deposits in urine, generally associated with either sciatica or herpes. I don’t think there is anything else I can tell you. These symptoms worry me a bit; and although I am as a rule very buoyant and easy-going, I fancy there is something that may develop into coarse brain disease. I told you that I thought sexual connection increased my symptoms. When I was first attacked, I used to syringe my ear, but it made me sick, and produced a stinging in my right eye. These symptoms I have told you of are very often, as they are *now* attended with a full heavy dull feeling in my head, and it seems to deaden my intellect, and render me powerless for consecutive thought. I am positive my memory is affected; and sometimes when I am conversing I can’t find the word in ordinary use that I want.”

## NOTE ON A REFLEX MECHANISM IN THE FIXATION OF THE EYEBALLS.

BY W. R. GOWERS, M.D.,

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THE phenomena of the fixation of the eyes by which, without conscious effort, they are kept directed to a given point, is attributed to a complex co-ordinating mechanism by which their movements are adjusted. The conditions of this fixation, however, suggest very strongly that its mechanism is not merely under the direction of a volitional centre, but that it is in part reflex. The motor impulses are constantly related to a sensory impression—that proceeding from the “fixing point” of the retina,—and analogy suggests that a reflex relation between this sensory impulse and the motor impulse may play an important part in the maintenance of the fixation. I am not aware that any recorded facts in physiology or pathology can be regarded as demonstrating the occurrence of such reflex action. The following observation, however, seems to constitute a proof of its existence.

A man who was lately under my care at the National Hospital for the Paralysed and Epileptic, on account of progressive muscular atrophy, with much rigidity, presented, during the last week of his life, the following symptom:—There was apparently a remarkable slowness in the movements of the eyes. If he was told to look at an object at a distance from that at which he was looking (of say  $45^{\circ}$ ), his eyes were kept for a moment fixed upon the object he had been looking at, and were then slowly moved until they were fixed on the desired point. On closer examination, however, it was seen

that this was not merely a slowness of muscular movement. The head was instantly turned towards the object he was told to look at, while the eyes were kept fixed on the first object, and they slowly followed the movement of the head, until they were again in mid-position and fixed on the required point.

It is evident that the maintenance of the fixation of the eyes, while the head was quickly moved, involved a movement of the eyeballs as rapid and considerable as the movement of the head, but in the opposite direction, so as to maintain the fixation of the globes. This movement was evidently independent of, and even opposed to, the will, and opposed to the voluntary movement of the head, with which that of the eyes is usually associated. Its source cannot therefore be found in an influence originating in the centres. It must be ascribed to an afferent impression, and the only afferent impression concerned was that visual impression originating from the fixing-point of the retina. The phenomenon seems thus to demonstrate the existence of a reflex mechanism maintaining the fixation of the eyeballs, and capable of doing so during movements of the head. Usually, this mechanism is in such strict subordination to the will, that its existence, apart from the voluntary control, cannot be recognised. In this case it was dissociated from the voluntary action by a resistance to the voluntary control of the reflex centre, and its existence was thus manifested.

It is evident that such a reflex action must be of the greatest importance in maintaining the fixation of the eyeballs during movements of the head, with the least possible expenditure of voluntary action. Probably the mechanism for this reflex is made functionally active during infancy by repeated visual impressions, repeated waves of impulse along the afferent tract. In the young infant no fixation of the eyeballs is to be observed, but after repeated visual impressions have occurred for several weeks, an intense impression leads to a reflex fixation; the child's eyes follow a light. The seat of the reflex process is probably the corpora quadrigemina.

The analogy between this reflex inertia of the eyeballs, as it may be termed, and the muscular fixation or rigidity in the limbs, which was very conspicuous in the same patient, is

obvious. The movements of the limbs were slow, just as that of the eyes, and just as the movements of the limbs are in paralysis agitans. The analogy suggests that such muscular rigidity is, in part at least, reflex (the afferent impression probably proceeding from the muscles themselves), and that there is a separation of the reflex centre from the voluntary centre, or at least a resistance to the action of the latter, which is only slowly overcome.

ON THE WEIGHT OF THE BRAIN AND ITS  
COMPONENT PARTS IN THE INSANE.

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*Lord Chancellor's Visitor.**(Continued from Vol. I., p. 518.)*

WHEN considering the relative weights of the two hemispheres of the brain, it occurred to me that their ordinary relations to each other in this respect might possibly be reversed by disease of long standing. It seemed not improbable that the cortical centres which are last organised, which are the most highly evolved and voluntary, and which are supposed to be located in the left side of the brain, might suffer first in insanity, which consists essentially in reduction from a higher voluntary to a lower automatic sphere. And not only did it seem probable that these centres would be first invaded, but also that they would be most deeply implicated, and so that the anatomical changes which correspond with mental disease would advance more rapidly and further in the left hemisphere, in which voluntary movements have a leading representation than in the right, in which their representation is perhaps subordinate to that of automatic movements. In order to test this supposition, I selected from my 400 cases 32 in which the insanity was unmistakably acute in character, and of recent origin, and in which therefore atrophic changes consequent upon it, might be presumed to be nearly or altogether absent. I speculated that in these cases, in which the cerebral lesion must have consisted in abnormal rather than defective nutrition, I should arrive as nearly as might be at the state of matters; as regards the relative weights



of the hemispheres, which exists during health. These cases are summarised in Table IV., and it will be seen that in them the right hemisphere is still the heavier of the two.

TABLE IV.

TABLE showing the WEIGHT of the BRAIN and its COMPONENT PARTS, in 32 cases of the Acute Forms of Insanity.

No.	Whole Brain.	Right Hemisphere.	Left Hemisphere.	Cerebellum.	Pons Varolii.	Medulla Oblongata.	No.
Average	Grammes.	Grammes.	Grammes.	Grammes.	Grammes.	Grammes.	Average.
	M. 1422·3	618·2	616·1	160·4	19·7	7·9	
	F. 1280·6	558·0	556·7	143·4	15·9	6·6	
	T. 1351·5	588·1	586·1	151·9	17·8	7·3	

Here the absence of atrophic changes is sufficiently evident, for the male brain in these 32 cases exceeds the male brain in the whole 400 cases by 87·6 grammes in weight, while the female brain presents an excess of 82·1 grammes. Whatever fallacy as to the weight of the hemispheres might therefore arise from these changes and their unequal distribution has now been got rid of, and yet we still find the right hemisphere ahead of the left one in weight. The differences between the two is 2·1 grammes for the male and 1·3 grammes for the female sex, and is not thus as great as that which separated them in the total number of brains examined, which was 3·7 for males and 2·1 for females. Nor is the proportion of cases in which the right hemisphere exceeded the left in weight as great as it was in the total number of cases, for in only 15 of the 32 cases was the right side of the brain the heavier. In 10 cases the left hemisphere was heavier than the right, and in 7 both were equal. Hence it might be urged that colour is given to the hypothesis that dextral pre-eminence in weight in the brain is a product of disease. But the superiority of the right hemisphere is still well marked, and the excess in weight of each hemisphere over that of the corresponding hemisphere, in the total number of cases, and in the 368 cases which remain after the 32 acute cases have been subtracted, is nearly the same in both.

TABLE V.

	Whole Brain.	Right Hemisphere.	Left Hemisphere.	Cerebellum.	Pons Varolii.	Medulla Oblongata.
	Grammes.	Grammes.	Grammes.	Grammes.	Grammes.	Grms.
Total Cases (400) .	1281·6	557·4	554·4	145·3	17·8	6·7
Acute Cases (32) .	1351·5	588·1	586·4	151·9	17·8	7·3
All others (368) .	1275·5	554·8	551·6	144·7	17·8	6·6
Excess of Weight in <i>Acute Cases</i> , as compared with all other cases (per cent.) . . }	6·0	6·0	6·3	5·0	0·0	10·6

The facts, as far as they are at present ascertained, appear to me to point to the conclusion that in a large majority of persons, sane and insane, the right cerebral hemisphere is slightly heavier than the left one. There is at any rate no warrant for the belief which has gained currency, and contributed to the construction of some neat theories that the reverse holds good, and that the left hemisphere is the leading one, in bulk and weight, as well as in the initiation of voluntary movements. As will be subsequently pointed out, there are grounds for supposing that the two halves of the brain differ materially in development in different regions, and that with the gradually increasing asymmetry of the convolutions, which keeps pace with intellectual growth in the individual and the race, there goes a gradually increasing variation in the relative weights of the hemisphere. In a few of the lower animals on which I have made observations, the cerebral hemispheres do not differ in weight to an extent at all approaching what is seen in the human subject.

In confirmation of the opinion which I expressed that the difference in weight of the male and female brain is really greater than would be inferred from observations made on chronic lunatics, I would call attention to the fact that the sex difference in the acute cases in Table IV. is considerably greater than it is in the whole number of cases in Table I.:

in the latter case it is 136·2 grammes, in the former it is 141·7 grammes.

The heaviest brain amongst the acute cases was that of J. C., a youth who died of exhaustion during acute mania, at the age of 15, and was reported to have been remarkable for his precocious intellect, and powers of acquisition; his brain weighed 1605 grammes. The lightest was that of a female aged 22, who sank under pneumonia and erysipelas while labouring under puerperal mania; her brain weighed only 1133 grammes.

The average weights of the cerebellum given in Table I., which are 151·4 grammes for the male and 135·7 for the female sex, agree closely with those given by Dr. Boyd in his Somerset Asylum tables, which are 150·3 grammes for the male sex and 137·1 for the female. This organ increases steadily in absolute weight, and in weight relative to the total weight of the brain (see Table II.) in each decennial period of life till between 40 and 50 in males and 30 and 40 in females; and it would thus seem that it is in each sex about 10 years later in reaching maturity than the cerebral hemispheres. It would also seem that the cerebellum does not participate to anything like the same extent as the cerebral hemispheres in the loss of substance that is commonly associated with mental diseases, for it is at its greatest weight at those very periods of life when the hemispheres are most reduced in weight by the degenerative changes to which they are liable. This comparative immunity of the cerebellum from simple and numerical atrophy may perhaps throw light upon the rather startling conclusion arrived at by Dr. Skae and adopted by Dr. Thurnam, that the average weight of the cerebellum is somewhat greater in the insane than in the sane. It is of course possible that a derangement in the normal relations subsisting between the great and little brain may constitute one of the conditions predisposing to insanity, but we cannot accept this proposition without more convincing evidence than has as yet been adduced in its support.

The cerebellum is, as will be seen a little later, only comparatively and not altogether exempt from the pathological changes that accompany insanity. It obviously undergoes senile decay in as marked a degree as the hemispheres.

The greatest weight to which the cerebellum attained in the 400 cases included in my tables was 182 grammes. This it did in three cases, all males: that of T. M., No. 374, who died of sclerosis of the brain; that of J. C., No. 318, who died of pneumonia interrupting acute mania; and that of G. O., No. 299, who died of general paralysis. The lightest male cerebellum was that of G. O., No. 188, an epileptic dement, who laboured under chronic hydrocephalus, and died in the *status convulsivus*, in whom it weighed 108 grammes. The heaviest cerebellum amongst the females was that of E. W., No. 242, who died of pneumonia intercurrent in acute mania and Bright's disease, and in whom it weighed 173 grammes; and the lightest was that of E. P. O., No. 93, who died of heart disease, and in whom it weighed 93 grammes. The range of variation in weight is therefore proportionately greater in the cerebellum than in the cerebrum.

The cerebellum bears the same proportion to the weight of the hemispheres in the two sexes, at all ages taken together, but at different decennial periods the proportion varies considerably in accordance with the rule already laid down, that the hinder brain is fully evolved ten years earlier in women than in men. In both sexes it attains its fullest development at the prime of life, but before the time at which the mental faculties have mounted to their utmost vigour and cultivation, and in both it keeps pace in its decay with the failure of the vital activities of the body.

The pons Varolii is exceedingly constant in weight, and after attaining its full size, which it does at about twenty years of age, it varies but little during the remainder of life. It is apparently invulnerable to senile decay, which so markedly reduces the bulk and substance of other parts of the encephalon, for in my observations it actually weighed more after seventy years of age than it did between thirty and forty. It is involved also, but to a trifling extent, in the various encephalic changes connected with insanity. On reference to Table IV. it will be observed that it weighed in the thirty-two acute cases comprised in that table precisely the same that it did in the whole 400 cases comprised in Table I.

The largest pons Varolii encountered was in a male general

paralytic, J. S., No. 132, forty-four years of age, in whom it weighed 30 grammes (it weighed 29 grammes in one case, and 28 in three); and the smallest was in a case of chronic disorganisation of the brain, with numerous small old clots in a female, H. C., No. 180, aged sixty-eight, in whom it weighed just 8 grammes.

The medulla oblongata is also subject to but little alteration in weight at the different epochs of life. It reaches its maximum between twenty and thirty years of age, and after sixty it dwindles to a slight degree. The largest medulla oblongata observed weighed 14 grammes, this weight being attained in nine cases of varied character, seven males and two females, and the smallest observed weighed 3·5 grammes, this weight being reported in three cases.

In Table VI. the 400 cases which have been summarised in previous tables are so sorted as to set forth, as distinctly as may be, the manner in which the weight of the brain is affected by the various morbid conditions associated with the different varieties of insanity. I am not now concerned to defend the system of classification adopted in this table, which is not exhaustive, because it embraces only those forms of insanity in which there was a fatal issue in one or more of the 400 cases which I have under consideration, and which is open to many objections, scientific and practical. I have made use of it because the cases with which I had to deal readily lent themselves to arrangement under it, and because I think it will be unambiguous to those who are engaged in the treatment of the insane.

From this table we learn that there is a marked diminution in brain-weight in those forms of insanity that are classified under states of mental weakness, and that this applies to both sexes, but is especially true of the male sex. The average weight of the brain in all the forms of mental weakness taken together is 1312·3 grammes for the male sex, 1172·8 for the female, and 1263 for both sexes. But the average weight of the brain in all the forms of mental exaltation taken together is 1379·3 for the male sex, 1242 for the female, and 1316·1 for both sexes; and in all the forms of mental depression taken

TABLE showing the AVERAGE WEIGHT of the BRAIN and its COMPONENT PARTS

Forms of Mental Disease.			Both Sexes.						
			Total Deaths.	Whole Brain.	Right Hemisphere.	Left Hemisphere.	Cerebellum.	Pons Varolii.	Medulla Oblongata.
States of Mental Weakness.	Amentia	Idiocy . . . . .	5	Grm. 1101·4	Grm. 489·2	Grm. 461·0	Grm. 129·6	Grm. 15·6	Grm. 6·0
		Imbecility . . . . .	4	1246·9	548·4	538·7	137·3	16·3	6·2
	Dementia	Simple and Acute Dementia	22	1315·1	568·3	575·2	146·6	18·3	6·7
		Consecutive and Chronic Dementia . . . . .	25	1265·4	552·3	542·8	145·6	18·4	6·3
		Organic Dementia . . . .	40	1261·3	547·4	547·3	142·8	17·4	6·4
		Paralytic Dementia . . . .	76	1225·4	526·9	523·8	149·6	18·1	7·0
		Epileptic Dementia . . . .	40	1330·2	583·2	582·7	140·5	17·2	6·6
		Alcoholic Dementia . . . .	1	1307·0	524·0	584·0	166·0	25·0	8·0
		Senile Dementia . . . . .	56	1260·5	549·9	544·6	141·4	18·1	6·5
States of Mental Exaltation.	Mania	Pyrexial Mania . . . . .	8	1310·7	571·8	570·9	143·9	16·9	7·2
		Simple Mania . . . . .	3	1370·1	593·0	595·4	156·0	16·3	9·4
		Mania à Potû . . . . .	1	1466·5	640·0	648·0	150·0	22·0	6·5
		Acute Mania . . . . .	16	1378·3	599·9	594·7	159·5	17·9	6·3
		Puerperal Mania . . . . .	3	1272·2	554·3	549·7	146·3	16·0	5·9
		Hysterical Mania . . . . .	1	1283·1	567·0	560·0	135·0	14·0	7·1
		Recurrent Mania . . . . .	5	1363·2	589·6	602·2	144·0	19·6	7·8
		Chronic Mania . . . . .	50	1288·1	562·5	557·3	144·3	17·4	6·6
	Monomania	Monomania of Pride . . . .	10	1359·8	593·6	588·2	153·4	18·3	6·3
		Monomania of Suspicion. . .	3	1191·7	518·7	513·3	137·3	17·0	5·4
States of Mental Depression.	Melancholia	Simple Melancholia . . . .	10	1303·2	572·9	562·1	143·1	18·2	6·9
		Acute Melancholia . . . . .	6	1355·7	587·2	589·8	150·3	19·6	8·8
		Delusional Melancholia . . .	12	1320·1	577·7	577·3	141·1	17·2	6·8
		Hypochondriacal Melancholia	3	1419·4	621·8	621·3	150·3	19·3	6·7

I.

400 Cases of both Sexes, classified under 23 Forms of Mental Disease.

Males.							Females.						
Males.	Whole Brain.	Right Hemisphere.	Left Hemisphere.	Cerebellum.	Pons Varolii.	Medulla Oblongata.	Deaths of Females.	Whole Brain.	Right Hemisphere.	Left Hemisphere.	Cerebellum.	Pons Varolii.	Medulla Oblongata.
	Grm.	Grm.	Grm.	Grm.	Grm.	Grm.		Grm.	Grm.	Grm.	Grm.	Grm.	Grm.
3	1156·0	517·6	491·7	126·7	14·0	6·0	2	1019·5	446·5	415·0	134·0	18·0	6·0
2	1282·1	563·5	552·0	143·5	16·5	6·6	2	1211·8	533·5	525·5	131·0	16·0	5·8
2	1406·5	605·1	616·1	158·6	19·8	6·9	10	1205·4	524·2	526·2	132·2	16·5	6·3
7	1315·3	576·2	562·1	151·1	19·5	6·4	8	1159·5	501·4	501·9	134·1	15·9	6·2
1	1310·4	568·9	566·5	149·1	19·1	6·8	19	1207·1	524·0	525·9	135·7	15·5	6·0
0	1262·2	544·0	540·1	152·3	18·7	7·1	16	1087·7	462·5	462·5	139·9	16·0	6·8
3	1396·1	613·6	610·2	147·5	17·9	6·9	14	1207·9	526·6	531·7	127·4	16·0	6·2
	1307·0	521·0	534·0	166·0	25·0	8·0	—	—	—	—	—	—	—
	1319·1	576·0	570·2	147·7	18·3	6·9	24	1182·4	515·2	510·4	133·1	17·8	5·9
	1440·1	623·5	620·0	166·5	19·5	10·6	6	1267·5	554·7	554·5	136·3	16·0	6·0
	1420·6	616·5	616·5	159·5	17·5	10·6	1	1269·1	546·0	553·0	149·0	14·0	7·1
	1466·5	640·0	648·0	150·0	22·0	6·5	—	—	—	—	—	—	—
	1406·3	612·8	605·8	162·6	18·8	6·3	6	1331·8	578·5	576·2	154·3	16·5	6·3
	—	—	—	—	—	—	3	1272·2	554·3	549·7	146·3	16·0	5·9
	—	—	—	—	—	—	1	1283·1	567·0	560·0	135·0	14·0	7·1
	1398·1	602·3	621·4	146·7	20·3	7·4	2	1310·8	570·5	573·5	140·0	18·5	8·3
	1372·3	599·0	594·1	153·7	18·4	7·1	23	1189·4	519·8	514·0	133·3	16·3	6·0
	1370·4	596·7	593·7	155·6	18·3	6·1	3	1335·2	586·4	575·3	148·3	18·3	6·9
	1196·1	519·5	511·5	142·0	18·5	4·6	1	1183·1	517·0	517·0	128·0	14·0	7·1
	1400·9	618·6	610·8	145·6	18·4	7·5	5	1205·6	527·2	513·4	140·6	18·0	6·4
	1414·8	614·3	616·4	155·3	20·3	8·5	2	1237·5	533·0	536·5	140·5	18·0	9·5
	1481·8	650·2	651·7	152·3	20·8	6·8	8	1239·2	541·4	540·0	135·5	15·4	6·9
	1419·4	621·8	621·3	150·3	19·3	6·7	—	—	—	—	—	—	—

together it is 1428·1 for the male sex, 1227·8 for the female, and 1331 for both sexes. And the contrast between states of mental weakness and those of mental exaltation and depression as regards the brain weights which are characteristic of them would be even greater than it is here represented to be, if two forms of insanity which are arranged under states of exaltation, viz. chronic mania and the monomania of suspicion, were placed in a separate division, or were transferred to states of mental weakness, which, for reasons to be hereafter mentioned, they might not improperly be. We should then have the following brain-weights for both sexes, corresponding with the three great divisions of mental diseases :

States of mental weakness . . . .	1263·0
„ „ exaltation . . . .	1344·1
„ „ depression . . . .	1331·2

It will be noticed that the weight here given for states of exaltation corresponds closely with that given for both sexes in the acute cases summarized in Table IV. Of course, a majority of these acute cases were instances of mental disease by exaltation, but a few instances of mental disease by depression were also included amongst the acute cases, as the point held chiefly in view in selecting these was not the specific character of the mental affection, but its speedy termination in death before necrobiotic changes had had time to interfere with the normal relations of the constituents of the encephalon. But in some of the cases classified under states of mental exaltation in Table VI., the mental disease had been of considerable duration, preserving, however, its essential characters to the last.

There is some difficulty in deciding whether the weight of the brain in states of mental exaltation, or that in states of mental depression should be received, as the nearest approach to the weight of the healthy brain. Doubtless in states of mental exaltation there is almost invariably hyperæmia active or mechanical of the whole brain, or of a segment of it, which is recognised after death as capillary or ramiform injection, and which must perceptibly augment the weight of the brain. But with the hyperæmia there goes increased tissue-waste and molecular disintegration, which must tend to



reduce the weight of the brain. The specific gravity of the blood, however, is considerably greater than that of the histological elements of the nerve centres, and the probability is that, as long as congestion lasts in the brain, its weight is slightly greater than it would be in a state of health. In states of mental depression, on the other hand, there is commonly anæmia, and some wasting consequent upon a deficient supply of suitable nutritive material. In acute melancholia, however, there is hyperæmia. Upon the whole, it would seem that the weight of the brain in states of mental depression is our best guide to the healthy standard of brain-weight. Perhaps that might be fixed at a point midway between the average weights for states of mental exaltation and depression.

Table VI. must convince us on the most cursory glance that insanity, taken as a whole, and when fatal, is associated with a diminished brain-weight. Under states of mental weakness there are marshalled (if chronic mania and the monomania of suspicion be included under these states) 322 of the whole 400 cases, and it is in these states of mental weakness that loss of brain-weight occurs. Under states of mental exaltation and depression, in which the brain-weight is maintained somewhere near the normal standard, the whole number of cases is only 78. We may dismiss, therefore, without further refutation, the statement of Parchappe that insanity has the effect of increasing the weight of the brain, and that of Skae that "the average weight of the brain is increased in persons dying insane." It may be taken as proved that insanity on the average of any considerable number of cases reduces brain-weight, and we have certainly no evidence to justify the theory that those who are attacked by insanity are of superior cerebral development to the rest of the community who escape mental disease. The multiplicity and diversity of the causes by which insanity is produced are of themselves sufficient to negative this theory.

Proceeding to analyse Table VI. a little more minutely, we perceive that the lowest brain-weights recorded are those connected with states of amentia, or original privation of the mental powers, in which it is not degeneration but arrested

growth of the brain, that is responsible for its inferiority in weight. The five idiots and imbeciles whose brains were examined were by no means of the lowest type, and hence the average brain-weight given under this heading is not to be taken as an average for the class.

The next lowest average brain-weight to that found in amentia, is discovered after a long and unexpected leap under the monomania of suspicion—a state of mental exaltation. But the three cases included under this heading in my table ought perhaps to have been transferred to that of alcoholic dementia, for in all of them those dangerous delusions of conspiracy and persecution, which are in certain persons the offspring of long-continued intemperance, had after a protracted course merged into a general deterioration of the faculties. In all of them, however, the delusions could be recognised until the end, which was immediately brought about, not by the cerebral changes but by pelvic abscess, pneumonia, and bronchitis, and hence their classification as monomania of suspicion. Only one case of alcoholic dementia is included in the table, and that not a peculiarly characteristic one; but a reference to notes of other cases convinces me that in this form of insanity the brain wasting is extreme.

Next to the monomania of suspicion, in ascending the scale of brain-weights comes paralytic dementia or general paralysis, in which, in both sexes, there is pronounced cerebral atrophy. The shrivelling of the cerebrum, and more particularly of the gyri of the frontal and parietal lobes in this malady is all the more remarkable, because the patients who die of it are mostly at the zenith of life, when the brain ought to be at its greatest weight. In the West Riding Asylum it is believed that as much as it was practicable to do, was done in the way of careful nursing and medical treatment to draw out the malady to its utmost possible extent; and thus no doubt brain wasting was carried in the cases included in this table to the furthest point, which is compatible even with vegetative life.

Immediately above paralytic dementia stands organic dementia in the scale of brain-weights, in which the whole brain was 1310·4 grammes in men, and 1207·1 in women. In this disease there is not only attenuation of the gyri, but

softening of the cerebral substance in the neighbourhood of old clots or embola, not large enough at once to extinguish life.

Consecutive and chronic dementia, a form or forms of mental disease, embracing so many of the inmates of our lunatic hospitals, whose nervous systems have been irreparably damaged by the acute storms of disease, or who have subsided quietly into the depths of fatuous degeneration, is represented in Table VI. by a brain-weight only a shade greater than that of organic dementia; the average for males being 1315·3 grammes, and for females 1159·5 grammes. Senile dementia, the result of atheromatous changes in the vessels without rupture, and clot, or the expression of the results of life-long wear and tear in nervous processes, has a brain-weight of 1319·1 grammes for males, and of 1182·4 for females. In both chronic and senile dementia, the loss of brain-weight is proportionately greater in the female than in the male sex.

Simple and acute dementia—two distinct but allied forms of mental disease, here for convenience grouped together—give an average brain-weight of 1406·5 grammes for males, and 1205·4 for females, that is to say, 1315·1 for both sexes. Here, therefore, the loss of brain-weight has been comparatively small. Under simple and acute dementia are arranged some of the mildest cases of mental impairment that find their way into a lunatic hospital, and some cases of sudden and sharp, but short-lived mental prostration.

Of all the forms of mental weakness, epileptic dementia has the greatest brain-weight in both sexes. Doubtless the ultimate tendency of epilepsy is to induce atrophy of the brain, which is seen when death takes place during epileptic stupor, although even then the wasting is by no means as advanced as it is in other forms of dementia; but primarily epilepsy tends to set up a state of induration or even hypertrophy of brain. Then in the cases of epilepsy that are cut short by accident, as by suffocation, or that terminate in a fit or in the *status epilepticus*, there is frequently great cerebral congestion, which adds materially to the weight of the brain when it is placed in the scales. A large proportion of asylum epileptics are in the most advanced stage of the disorder, and die in the state of

stupor, so that the brain-weights given in Table VI. must not be taken as criteria for those of epileptics in general.

Amongst the states of mental exaltation, the brain-weight is high in pyrexial, simple, and acute mania, and low in chronic mania. In the last-named form of mental disease, a primary acute attack of madness, and subsequent mental excitement and disorder of long continuance, extending often over many years, have unquestionably deteriorated the brain in all respects, and diminished its weight. Chronic mania tends, in the course of time, to merge into chronic dementia, and I ought perhaps to have followed Griesenger and grouped it with states of mental weakness. A certain limited persistence of spontaneity and mental and bodily activity, in the cases classified as chronic mania, and the absence of the progressive degeneration which is characteristic of dementia, induced me to retain these cases amongst states of mental exaltation, to which they all indisputably at their outset belonged. In arranging my cases under the different forms of mental disease enumerated in Table VI., I have not trusted to anything so treacherous as entries in Registers, returns to Commissioners, or diagnoses made at the time when the patient was brought under care. In each case, I have reviewed its whole history, and have classified it in accordance with that, giving particular regard to the symptoms presented at the time of death.

In recurrent mania and the monomania of pride, there is evidence of slight brain-wasting in the male, but of none in the female sex. In the single case of mania à potû, the patient, who was naturally possessed of a good brain, died during delirium tremens, and his nerve-centres were found much congested.

It is in states of mental depression, as I have already argued, that the brain is found in a condition most nearly approaching to normal integrity. Melancholia is, in its milder forms, a mere functional disturbance, and even when it assumes a severer type, and is connected with structural changes in the great nerve-centres, these are not of a severity at all comparable with what occurs in states of mental weakness or exaltation. When death happens in melancholia it is but rarely that it is attributable to any encephalic condition. Almost invariably

it is due to visceral or constitutional diseases that are only indirectly dependent upon the primary neurosis and that do not seriously interfere with cerebral nutrition. Thus in only one of the thirty-one cases of death during melancholia, included in Table VI. was brain disease the cause of death. Phthisis was the cause of death in five of these cases; pneumonia in four; bronchitis in three; pleurisy in four; heart disease in three; Bright's disease in four; and general tuberculosis, ulceration of the intestines, cancer of the breast, cancer of the uterus, pyæmia, erysipelas, and diarrhœa in one each; and it is certainly remarkable that in sixteen out of thirty-one cases of melancholia, disease of the lungs and its covering should have been responsible for a fatal termination. But the diseases enumerated, whether of the lungs or other organs, produce comparatively little wasting of the cerebrum and its appendages, and hence the brains of melancholics, of all brains examined in the post-mortem room of a lunatic hospital, most closely resemble the healthy brain in all respects. There may be slight wasting or slight engorgement in acute melancholia; there may be intense injection of the grey matter of the hemispheres in certain areas; but as a rule, the encephalic structures do not present those coarse alterations which are so obvious in demented and maniacs. Bearing this in mind, it is instructive to notice that in the brain of melancholics included in Table VI., the sex difference in weight is much more pronounced than in the brains of lunatics who suffered from other varieties of insanity. In melancholics this difference amounts to  $200\cdot3$ , whereas in the whole number of the brains weighed it was  $136\cdot2$  grammes.

The fact that the brains of melancholics, which of all lunatic brains are most like those of sound-minded people, exhibit the largest ratio of sex difference, is certainly corroborative of the view taken in an earlier part of this paper, that insanity tends to level down the sex difference, and that the male brain on an average exceeds the female brain in weight in an amount considerably greater than these estimates which have been formed upon lunatic asylum statistics would lead us to believe.

The only point in connection with the differences in brain-

weight in the sexes in the various forms of mental disease which it is important to notice is, that throughout the states of mental weakness, the loss of weight sustained by the brain in the female sex is vastly less than that which it suffers in the male sex. The difference between the greatest and the least brain-weight recorded in Table VI., exclusive of states of amentia, is 270·4 grammes for the male sex and 172·3 for the female, a fact which indicates the much greater severity of the pathological vicissitudes to which the male organ is exposed.

The number of cases falling to each particular form of mental disease in Table VI. is so small that no profit could accrue from arranging them with reference to age at the time of death.

As regards the relative weights of the cerebral hemispheres in the twenty-three forms of mental disease enumerated, the following observations may be made, with the qualification that the number of cases is too minute to warrant anything more than a mere suggestion in each instance.

1. In states of amentia the right is heavier than the left cerebral hemisphere in both sexes.

2. In senile dementia the right hemisphere preponderates in both sexes.

3. In simple and acute dementia the left hemisphere preponderates in both sexes.

4. In paralytic and epileptic dementia the right hemisphere is heavier than the left one in the male, while in the female sex they are nearly equal.

5. In chronic mania the right hemisphere is markedly heavier than the left in both sexes.

In the dementia of general paralysis the cerebellum does not share to anything like a full extent in the wasting by which the cerebrum is so seriously reduced; indeed, the cerebellum is less wasted in the dementia of general paralysis than in any of the other chronic forms of dementia, while the cerebrum is more wasted than in any other form of mental disease without exception. The average weight of the cerebellum in general paralysis is 152·3 grammes in males, and 139·9 in females, while the average weight at all ages for all the 400 cases was 151·4 for males, and 137·7 for females. What may be the significance of this comparative immunity of the

cerebellum from the destructive changes which so rapidly and extensively denude and degrade the cerebrum in this malady, we are not yet in a position to suggest.

In striking contrast with the weight of the cerebellum in general paralysis is its weight in epilepsy; there the cerelet is of inordinately small weight in both sexes, while the cerebrum is of great weight in comparison with its weight in the other chronic forms of dementia. That there is often wasting of the cerebellum in epilepsy is unquestionable; but whether the whole of the deficiency of weight in this organ in epileptics, which amounts to 11·4 grammes for both sexes short of the average weight recorded for acute cases, is to be ascribed to wasting, or whether some of it may not be attributable to arrested development, I am not prepared to say.

In acute mania in both sexes the cerebellum is of great weight, absolutely and relatively to the weight of the hemispheres; and, indeed in almost all the forms of mental exaltation and depression the weight of the organ contrasts notably with what is seen in states of mental weakness.

But little variation is observable in the weight of the pons Varolii and medulla oblongata in the several varieties of mental disease.

Of the 400 patients whose brains I examined, twenty-five males and nineteen females, forty-four in all, died of phthisis pulmonalis. The weights of their brains, separated from the others, and subjected to calculation, give the results shown in Table VII.

TABLE VII.

TABLE showing the WEIGHT of the BRAIN and its COMPONENT PARTS in 44 Cases of Phthisis (25 Males and 19 Females).

No.	Age.	Whole Brain.	Right Hemisphere.	Left Hemisphere.	Cerebellum.	Pons Varolii.	Medulla Oblongata.
		Grammes.	Grammes.	Grammes.	Grammes.	Grammes.	Grammes.
Average	(Males .	1372·0	599·5	598·1	149·6	17·9	6·9
	(Females	1234·5	535·8	538·7	136·8	16·9	6·3
	(Total .	1312·6	572·0	572·4	144·1	17·5	6·6

It appears that the brains of the phthisical are wasted to a certain extent. They fall short in weight of the brains in acute cases to the extent of 50·3 grammes in the male and 46·1 in the female sex, but they transcend the general average of the whole 400 cases to the extent of 24·3 grammes for the male, and 26 for the female sex. The forms of insanity from which these phthisical patients suffered was perhaps more instrumental in determining the brain-weight than their diathesis or lung disease. Eighteen of the forty-four laboured under chronic mania, six under epileptic dementia, six under chronic dementia, five under mania (simple, acute, and puerperal), four under melancholia, three under idiocy and imbecility, and two under delusional insanity.

In carrying out my researches as to brain-weights, it soon became evident that in tracing the relations of the size and weight of the cerebrum to the intellectual power of the race, or of the individual in health and disease, it was necessary to obtain information as to the relative weights of the lobes of which each hemisphere of the brain is composed. On this subject no information has hitherto been published, so that I had to undertake an original investigation in order to obtain it. This I did in 1875, extending my observations on the brains which I examined to the weight of the lobes, as well as of the hemispheres. My observations were, however, broken off when I had weighed the lobes separately in only sixty brains. The results obtained from these sixty brains are here set forth, not as supplying any definite standards of comparison, but simply as furnishing certain general indications that are not without value and significance.

The weighing of the cerebral lobes was conducted as follows:—

The brain having been denuded of its pia mater, and the hemispheres having been separated and weighed in the manner already described, each of them was laid on its flat inner surface on a board, and was treated in the following manner. The ascending frontal and ascending parietal gyri were carefully separated from each other by the fingers of the left hand of the operator, and a large and long brain-knife was introduced between them, and being held perpendicular to the



board and in the line of the sulcus, was made by one clean cut to separate the frontal lobe from the rest of the brain. The remainder of the brain being then turned over, a second incision was made in the line of the internal parieto-occipital fissure, up to the part where it is crossed by the calcarine fissure, in the line of which the third incision was made, so separating from the cerebral mass a portion which might be roughly taken to correspond with the occipital lobe. The remainder of the brain being again turned over, the last cut was made in the line of the horizontal limb of the Sylvian fissure, and severed the temporo-sphenoidal from the parietal lobe.

It need not be said that this was a somewhat unsatisfactory method of partitioning the brain, and was adopted simply because none better could be devised. The lobes of the brain are not separated from each other by scientific frontiers, but by arbitrary lines, and the existence of secondary gyri often renders it difficult to trace out their boundaries with sufficient exactness. Great care was, however, taken to pursue precisely the same method of subdivision in each case. It might have been more satisfactory had I followed Gratiolet, and made the ascending limb of the Sylvian fissure the posterior boundary of the frontal lobe, for more of Ferrier's motor region would then have been included in the parietal lobe. But the fissure of Rôlando is less apt to be interrupted by secondary gyri than the ascending limb of the Sylvian fissure, and forms therefore a better line of division; and whatever may be the functional relations of the ascending frontal gyrus, it is certainly in intimate morphological connection with the three tiers of frontal gyri, as is evident in the foetal brain, and in the brains of the Simiæ, while it is sharply marked off from the ascending parietal gyrus. I think some interesting results would be obtained by weighing separately, in a large number of cases, sections of the cerebrum separated from each other, not in the lines of sulci, but in straight lines, at fixed distances from given points.

Tables VIII. and IX. (pp. 60, 61) exhibit the general results of my weighings of the cerebral lobes:—

TABLE VIII.

TABLE showing the AVERAGE WEIGHT of the BRAIN and its COMPONENT PARTS, in 60 Cases, of both Sexes;  
31 Males and 29 Females.

	Whole Brain.	Right Hemisphere.					Left Hemisphere.					Cerebellum.	Pons Varolii.	Medulla Oblongata.
		Total of Right Hemisphere.	Right Frontal Lobe.	Right Parietal Lobe.	Right Temporal Sphenoidal Lobe.	Right Occipital Lobe.	Total of Left Hemisphere.	Left Frontal Lobe.	Left Parietal Lobe.	Left Temporal Sphenoidal Lobe.	Left Occipital Lobe.			
Males . .	Grm. 1329·3	Grm. 581·5	Grm. 245·7	Grm. 131·8	Grm. 131·7	Grm. 72·3	Grm. 576·5	Grm. 238·3	Grm. 139·9	Grm. 132·0	Grm. 66·3	Grm. 147·2	Grm. 17·5	Grm. 6·6
Females .	1183·5	510·7	217·3	116·3	114·8	62·3	513·7	212·5	121·1	118·8	61·3	137·2	15·9	6·0
Total . .	1258·8	547·3	232·0	124·3	123·5	67·5	546·1	225·8	130·8	125·6	63·9	142·4	16·7	6·3

TABLE IX.

TABLE showing the RELATIVE WEIGHTS of the COMPONENT PARTS of the BRAIN to the total Brain-weight  
(assumed to be 1000).

Averages of 60 Cases of both sexes; 31 males and 29 females.

	Whole Brain.	Right Hemisphere.					Left Hemisphere.					Cerebellum.	Pons Varolii.	Medulla Oblongata.
		Total of Right Hemisphere.	Right Frontal Lobe.	Right Parietal Lobe.	Right Temporal Sphenoidal Lobe.	Right Occipital Lobe.	Total of Left Hemisphere.	Left Frontal Lobe.	Left Parietal Lobe.	Left Temporal Sphenoidal Lobe.	Left Occipital Lobe.			
Males . .	1000·0	437·4	184·8	99·1	99·1	54·4	433·7	179·3	105·2	99·3	49·9	110·7	13·2	5·0
Females .	1000·0	431·5	183·6	98·3	97·0	52·6	434·1	179·6	102·3	100·4	51·8	115·9	13·4	5·1
Total . .	1000·0	434·7	184·3	98·7	98·1	53·6	433·9	179·4	103·9	99·8	50·8	113·1	13·3	5·0

The first point to be noted in Table VIII, is that the weight of the whole brain in the sixty cases included in it is considerably less than that of the whole brain in the 400 cases which were dealt with in the earlier part of this paper. In the 400 brains the average weight of the whole in both sexes was 1281·6 grammes, whereas in the sixty cases it was 1258·8, showing a deficiency of 22·8 grammes in the latter. This discrepancy is accounted for by the fact above mentioned, that the cerebral hemispheres, which were divided into lobes, were entirely stripped of their pia mater. The removal of this membrane and the escape of fluid from the surface of the cerebrum during its removal reduced the brain-weight in the sixty cases in which the lobes were weighed to the amount indicated. The loss of weight, as was inevitable under such circumstances, was distributed over all parts of the brain, and occurred in both sexes, though from some cause, which is not clear, it was much more marked in the brains of females than in those of males.

The second point deserving attention is the large relative weight of the frontal lobes, which make up about two-fifths of the weight of each hemisphere. Of course these lobes include the islands of Reil, which may be regarded as the starting-points of the frontal convolutions, and which it is impossible to dissect away from surrounding parts in such a manner that they might be weighed separately with any approach to trustworthy results. As the frontal lobes in man may be regarded as generally homologous with those regions at the anterior extremities of the hemispheres in the monkey which give negative results when subjected to electrical irritation, and with those small cerebral masses which in the dog and cat lie in front of the anterior limb of the sigmoid gyrus, it is evident that they attain in the human subject to a degree of development that must be pronounced enormous.

The third noteworthy point is that the frontal lobes in both sexes bear about the same proportion in weight to the other lobes of the cerebrum. Preconceived ideas might have led us to anticipate that these lobes would be found to be lighter in proportion to the rest of the hemispheres in women than in men; but this is not so. It may be that they are in some

respects less highly developed, that they are less richly convoluted, that their grey matter is shallower, that their ganglion-cells are less freely branched, or that their blood supply is less copious; but this, at any rate, is tolerably clear, that these lobes are not inferior in weight, in proportion to the weight of the brain as a whole, in women, to what they are in men. This is clearly indicated in the following table, which exhibits the average relative weights of the frontal lobes as contrasted with the weights of the other lobes in each hemisphere, the whole weight of each hemisphere being assumed to be 1·000 grammes.

TABLE X.

TABLE showing the PROPORTION of the FRONTAL LOBES, and of the remaining LOBES, to the total weight of the Right and Left Hemispheres respectively (assumed to be 1·000).

	Right Hemisphere.			Left Hemisphere.			
	Total of Right Hemisphere (assumed to be 1·000).	Proportion of Right Frontal Lobe.	Proportion of the Remaining Right Lobes.	Total of Left Hemisphere (assumed to be 1·000).	Proportion of Left Frontal Lobe.	Proportion of the Remaining Left Lobes.	
Males .	1·000	·423	·577	1·000	·413	·587	Males
Females	1·000	·426	·574	1·000	·414	·586	Females
Total .	1·000	·424	·576	1·000	·414	·586	Total

Among males the proportion that the right frontal lobe bears to the sum of the three other right lobes is as 423 to 577; the right frontal lobe is therefore equal to ·423 of the total weight of the right hemisphere. Similarly the left frontal lobe is to the sum of the three other left lobes as 413 to 587, and is equal to ·413 of the total weight of the left hemisphere. Among females the proportion that the right frontal lobe bears to the sum of the other right lobes is as 426 to 574; the right frontal lobe is therefore equal to ·426 of the total weight of the right hemisphere. Similarly, the left frontal lobe is to the sum of the three other left lobes as 414 is to 586,

and is equal to  $\cdot 414$  of the total weight of the left hemispheres. The share taken by the frontal lobes in the composition of the cerebrum would seem, therefore, to be slightly greater in females than in males; but if allowance be made for an excess in the number of cases of general paralysis and organic disease of the brain, in which the frontal lobes are invariably much wasted, amongst the thirty-one males included in the sixty cases upon which the table is founded, it may, I think, be fairly said, that the frontal lobes hold an equal proportion in weight to the other lobes of the brain, and to the whole weight of the hemispheres in both sexes.

A third point calling for remark is, that in both sexes the parietal and temporo-sphenoidal lobes were heavier on the left than on the right side, while in both sexes the frontal and occipital lobes were heavier on the right than on the left side. The sum of the left parietal and temporo-sphenoidal lobes exceeded the sum of the same lobes on the right side by  $9\cdot 8$  grammes in the male and by  $8\cdot 8$  grammes in the female sex, while the sum of the right frontal and occipital lobes exceeded that of the same lobes on the left side by  $13\cdot 4$  grammes in the male and by  $5\cdot 8$  in the female sex. The preponderance of the left parietal and temporo-sphenoidal lobes over the corresponding lobes of the other side in the female sex, is so much greater than the preponderance of the right over the left frontal and occipital lobes, that the relation of the two hemispheres is affected, the left being in the cases comprised in this table actually 3 grammes heavier than the right. In the male sex, however, the preponderance of the right frontal and occipital lobes is more than sufficient to counterbalance the preponderance of the left parietal and temporo-sphenoidal, so that the right hemisphere remains 5 grammes heavier than its fellow of the opposite side. In this connection it is important to observe, that in the 400 cases in which the hemispheres were weighed, the preponderance of the right over the left hemisphere was much less decided in the female than in the male sex, having been only  $1\cdot 8$  grammes in the former against  $2\cdot 8$  in the latter; and it may, perhaps, be suggested that this was due to the fact that the left parietal and temporo-sphenoidal lobes exceed those of the left side in a greater degree, while the

right frontal and occipital lobes exceed their fellows of the opposite side to a less degree in women than in men. It would appear that the tendency to symmetry in the two halves of the cerebrum is stronger in women than in men.

The pronounced excess in weight of the left over the right parietal lobe in both sexes, amounting in men to 8.1 grammes, and in women to 4.8 grammes, is very significant. These lobes, as weighed in my observations, include a large part of Ferrier's motor areas; and it is therefore interesting to find the lobe of the left side, in which the various volitional motor acts are supposed to have a leading representation, surpassing that of the right side so unmistakably in development. The muscles of the right side of the body being principally concerned in voluntary motor performances, we should expect the motor centres of the left hemisphere, which are more especially the organic basis of motor acquisitions, to be more highly educated and developed than the corresponding centre of the opposite side. And the results to which attention has just been directed are in harmony with this expectation. Then, again, in men who are engaged in handicrafts requiring skill and dexterity to a much greater extent than women, and in whom righthandedness is, I believe, more marked in degree, we should expect the motor centres of the left hemisphere to be more highly developed than in women. And again the figures confirm our preconceptions; for the left parietal lobe exceeds the right one by 8.1 grammes in weight in men, and only by 2.8 grammes in women.

Had a different system of partitioning the brain to that which I adopted been pursued, perhaps even more striking results would have been obtained, and certainly more trustworthy data would have been collected bearing upon the researches of Hitzig and Ferrier. In my observations, a large portion of the motor tract, the ascending frontal, and the posterior ends of the three tiers of frontal gyri were included in the frontal lobes. I am confident that a sufficiently extended series of brain-weighings, after a carefully designed scheme, will conduct to valuable conclusions. It is practicable for the unaided eye to discover in a majority of brains a distinct difference in Broca's convolution on the two sides; a bulging and fulness

TABLE XI.

TABLE showing the AVERAGE WEIGHT of the BRAIN and its COMPONENT PARTS, in 60 Cases, of both Sexes, at different Ages.

Ages.	Whole Brain.	Right Hemisphere.			Left Hemisphere.			Cerebellum, Pons Varolii, and Medulla Oblongata.
		Total of Right Hemisphere.	Right Frontal Lobe.	R. Parietal, R. Temp. Sph., and R. Occipital Lobes.	Total of Left Hemisphere.	Left Frontal Lobe.	L. Parietal, L. Temp. Sph., and L. Occipital Lobes.	
	Grammes.	Grammes.	Grammes.	Grammes.	Grammes.	Grammes.	Grammes.	Grammes.
All Ages	{ M. 1329·3	581·5	245·7	335·8	576·5	238·3	338·2	171·3
	{ F. 1183·5	510·7	217·3	293·4	513·7	212·5	301·2	159·1
	{ T. 1258·8	547·3	232·0	315·3	546·1	225·8	320·3	165·4
Under 20	{ M. 1149·0	534·0	228·0	306·0	481·0	199·0	282·0	134·0
	{ F. ———	———	———	———	———	———	———	———
	{ T. ———	———	———	———	———	———	———	———
20-30.	{ M. 1408·4	616·0	255·3	360·7	616·9	246·2	370·7	175·5
	{ F. 1160·5	504·0	233·0	271·0	492·0	201·0	291·0	164·5
	{ T. 1372·9	600·0	252·1	347·9	599·0	239·7	359·3	173·9
30-40.	{ M. 1328·3	572·0	237·7	334·3	578·7	238·5	340·2	177·6
	{ F. 1230·6	531·3	225·6	305·7	535·4	213·9	321·5	163·9
	{ T. 1266·1	546·1	230·0	316·1	551·2	222·8	328·4	168·8
40-50.	{ M. 1296·8	564·8	240·3	324·5	564·4	235·0	329·4	167·6
	{ F. 1131·5	486·7	215·0	271·7	486·3	214·0	272·3	158·5
	{ T. 1251·6	543·4	233·4	310·0	543·1	229·3	313·8	165·1
50-60.	{ M. 1317·3	574·4	243·0	331·4	567·0	234·2	332·8	175·9
	{ F. 1161·7	494·9	208·3	286·6	504·3	210·9	293·4	162·5
	{ T. 1217·3	523·3	220·7	302·6	526·7	219·2	307·5	167·3
60-70.	{ M. 1349·2	595·5	255·5	340·0	579·5	246·8	332·7	174·2
	{ F. 1181·3	515·9	216·3	299·6	514·4	213·0	301·4	151·0
	{ T. 1242·3	544·8	230·5	314·3	538·1	225·3	312·8	159·4
70 and upwards.	{ M. 1313·5	579·3	249·0	330·3	568·7	239·3	329·4	165·5
	{ F. 1213·7	530·5	227·0	303·5	529·0	216·5	312·5	154·2
	{ T. 1273·6	559·8	240·2	319·6	552·8	230·2	322·6	161·0



of the hinder part of the third frontal convolution on the left side, which has no counterpart on the right; and I cannot doubt that differences of a very significant character, and appreciable by the scales, would be revealed in various regions of the two hemispheres.

In two cases in which the right frontal lobe was heavier than the left, I have unfortunately been unable to ascertain whether the patient had been left-handed.

In Table XI. the weight of the frontal lobes is contrasted with that of the sum of the other lobes at different ages; but the number of cases available for arrangement in the several decades is far too small to enable any trustworthy average to be struck. The table suggests, however, that the relation as regards weight between the frontal and other lobes is maintained without material alteration throughout adult life and old age.

## Critical Digests and Notices of Books.

*The Hystero-neuroses.* By GEORGE J. ENGELMANN, M.D. St. Louis, M.O. Reprint from Vol. II. of 'Gynæcological Transactions,' 1878.

HYSTERO-NEUROSES is the name given by the author of the above paper to a series of nervous phenomena which occur in different parts of the body as a result of uterine irritation. Morbid conditions of the womb, both functional and structural, he maintains, cause such primary local excitement, as by reflex action induce secondary perverted function in various distant parts. The organs thus affected are not structurally changed, and the symptoms disappear on the cessation of the uterine disorder. The subjects of such afflictions are not as a rule hysterical, and the phenomena are not to be looked upon as belonging to that disease. It is pointed out, for example, that there is a close relation between psychological changes and disorders of the womb. It is stated that, in a large majority of women mentally affected, there are menstrual irregularities. Cases are given where epilepsy originated from cervical erosions, and which recovered after this disease was removed. In the same way melancholia, hypochondriasis, and other forms of insanity were caused by uterine displacements and disease, and they disappeared when these last were cured. The author further proceeds to show that affections of the eye, pharynx, larynx, bronchi, breasts, intestines and joints, may in the same way be associated with uterine disease, which he maintains causes these disorders entirely by reflex irritation. He enters more particularly into the effects produced on the stomach through this agency. He points out how common the symptom of dyspepsia is in those persons

suffering from disease of the womb, who are menstruating, or who are pregnant, and draws from this the conclusion that this is due to the effects of uterine irritation.

Dr. Engelmann's paper deals with a most important and interesting subject, with the nature of which we are as yet little acquainted, and a question which at the present time is attracting much attention amongst those working at nervous physiology and pathology. In criticising the views of the author, it must be at once conceded that his statements are to a certain extent probably correct, that many of the reflex phenomena which occur in women may have for their origin some irritation of the uterus, and that when this excitement is removed the nervous symptoms cease. The question, however, arises, is the uterus *per se* the only cause which may give rise to the spasms, convulsions, &c., which are said to be produced through its influence? We venture to doubt such a theory. Irritation to any peripheral sensory nerve may under certain circumstances induce reflex acts which manifest themselves by a variety of nervous phenomena in different parts of the body. An injury to the finger, for example, may result in tetanus or epilepsy; from teething there follow convulsions; worms in the intestines give rise to the same; renal, vesical, and hepatic calculi originate both motor and sensory disturbances. It is therefore not remarkable that an irritation of so important an organ as the uterus should be the starting-point of nervous reflex symptoms. Nay, considering how liable the womb is to functional as well as structural disorder, how important a part it plays in the psychical as well as in the physical well-being of women, it may be admitted that of all the nervous affections which follow peripheral irritation the majority arise from this source, simply because the uterus is perhaps the most important organ in the female economy, and the one which is the most frequently diseased. But we question whether uterine irritation in its effects differs in other respects from excitement of any other sensitive nerves, or that it induces any special or characteristic symptoms. An inquiry describing the reflex neuroses arising from worms, from teething, and so on, would be quite as justifiable as the one before us. In all, the result would most

probably be almost identical, the symptoms being modified only by the age, sex, and other conditions under which the patient might be placed. The physiology and pathology would in all be the same.

Dr. Engelmann states that he does not consider that the affections he describes and his views of their origin have received much attention in works on the subject. It must, however, be remarked that the symptoms themselves have been known and described from the earliest ages under the name of *Hysteria*, and precisely the same explanation given of their origin. Until recently, and even by some at the present day, the disease vaguely denominated *hysteria* was believed to originate in irritation of the uterus, and hence the name. Most pathologists, however, do not now hold so exclusive a doctrine, and recognise an independent origin of this malady. Whatever the real nature of this may be, all have long been acquainted with its symptoms, and the author, therefore, confirms the experience of ages as to facts, and explains them as *Actius* and his followers did centuries ago. In short, he gives the new name of *hystero-neuroses* to a series of phenomena which have been long recognised under the term *hysteria*.

Dr. Engelmann draws a distinction between these *hystero-neuroses* and *hysteria* as generally understood, pointing out that the patients who suffer from the symptoms described are not of necessity nervous or impressionable, and that the convulsions, &c., are purely reflex in character. He would therefore lead us to infer that the persons thus affected were otherwise healthy until the *neuroses* were developed by the uterine irritation. We, however, venture to question also this hypothesis. We believe that the uterine irritation is only the exciting cause of the motor and sensory reflex acts, and that a prior predisposition to nervous disease existed. All children during dentition do not have convulsions, spasms, &c., only a certain minority being thus attacked, and these probably have an irritable and susceptible nervous system, either inherited or acquired, which renders them not only more sensitive to slight irritations, but causes an exaggerated development of reflex results. Hence the eruption of a tooth in them produces convulsions, while in the healthy

child it is attended with little or no inconvenience. That such a nervous predisposition exists apart from the teething is further indicated by the fact that those children who are thus upset during dentition are in after life frequently attacked with chorea, epilepsy, hysteria, and other nervous diseases, showing that from the first they were the subjects of a nervous diathesis. Again, if teething alone caused convulsions it would be general, while we know it is comparatively the exception.

Precisely in the same way all and perhaps the majority of women with uterine affections do not suffer from spasms, hysteria, and other hysteroneuroses; on the contrary, it is notorious that gynæcologists tell us that subjects suffering from cancer and other grave affections of the womb are not by any means especially liable to nervous disorders. On the other hand, given a nervous instability, a morbid susceptibility to explosions of movement and pain, an irritation of the uterus, however slight, may be the spark which kindles up general nervous demonstrations. We see this frequently in those cases of greatly impaired health, and where there are the most severe nervous attacks, which are associated with the mildest forms of uterine disorder, with menstrual irregularities, and so on. If the general nervous system of a woman be healthy, we greatly doubt whether any disorder of the uterus would induce any of the more serious reflex symptoms described under the term hysteroneurosis; but given a delicate and highly nervous and impressionable patient, the slightest disturbance of the uterus or of any other organ might induce the severest form of reflex nervous disorder. If this be so, we are practically dealing with what has hitherto been named Hysteria, by which term has been understood a variety of neuroses occurring in certain peculiar constitutions, of the nature and origin of which at the present time we know very little. The expression of the author, hysteroneuroses, are simply those symptoms which arise in that disease from irritation of the uterus as an existing cause, in distinction to irritation from any other part of the body. We might therefore with equal reason speak of dental, cutaneous, or cerebral neuroses, according to the organ which originated the irritation.

It is often stated that if reflex phenomena are not so common with structural disease of the uterus, that they are specially liable to arise from functional disorder of that organ. The question then arises, Are these affections of the womb the veritable cause of the nervous disorders, or are they not the results? It is very generally assumed when hysteria and other nervous phenomena are associated with those uterine affections that the latter are the direct or indirect cause of the former. There is no evidence of this, on the contrary it appears to us that in the majority of cases the state of the nervous system is the cause and not the result of their functional disturbances. We believe that the amenorrhœa, for example, in delicate, impressionable young women is one of the symptoms of a general nervous disposition; and when we inquire into the past history of the patient, we often find that she has suffered from all the nervous symptoms before puberty, that she has probably inherited a neurotic tendency, and possibly had dental convulsions, chorea, or other such affections in her youth. On growing older the symptoms are modified by a variety of circumstances, and new ones are developed, among others, disordered menstruation. In such a case as this, which is so common, it would be as erroneous pathology to say that the amenorrhœa was the cause of any nervous symptoms which might subsequently arise, as it would be dangerous treatment to centre the attention on the uterine irregularities when the general health was so obviously at fault. We do not of course mean to deny, if tangible and removable disease of the womb exists, which can be shown to be the exciting cause of reflex action, that it should not be treated; but we suspect that such cases are not common, and that the functional uterine disorder is by far more often the effect than the cause of nervous disease.

A. HUGHES BENNETT, M.D.

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*On Regressive Paralysis* (Infantile Paralysis, Spinal Paralysis of Adults). By WILLIAM H. BARLOW, M.D., Consulting Physician to the Dispensary for Sick Children, Manchester. Manchester, 1878.

THE author of this reprint fears that as his work so soon follows the publication of Dr. Althaus's Essay on the same subject, he may be assumed to have borrowed some of his conclusions from the earlier published work.

But a very cursory glance at the paper will show that the clinical part of it represents a considerable amount of patient and conscientious work, and the analysis of sixty-three cases given quite irrespective of the results of treatment obtained, imparts a solid value to it. Concerning the style of the reports, we will only say that we trust in a future edition the author will give us more detail, if possible, on the progress of his cases, and the frequency with which his therapeutic measures were used, and will also eliminate the prolix account of the family history, which in the disease described appears to be useless.

Under the title "*Regressive Paralysis*," the author proposes to include cases hitherto known as essential paralysis of children and allied cases occurring in adults. There is something to be said for a revision of terms. Since the morbid anatomy of the infantile cases has been studied microscopically, disease of one or other of the anterior horns of the grey matter of the spinal cord has been always found.

Whether this lesion be primary or secondary, its discovery ought to exclude from our nosology the term "essential" as applied to such cases, because of its giving an erroneous view of the nature of the disease. The term "*infantile paralysis*" is unsatisfactory, because, first, paralysis occurring in infancy is not limited to this type; second, because although the most frequent period for the onset of this disease is during the first dentition, yet cases are met with in children beyond this period—for example, I have seen the disease beginning in a child aged  $7\frac{1}{2}$  years. But the strongest objection to the term is that a number of cases have been recorded of disease in adults having a nearly identical clinical behaviour.

Until some more acute fatal cases have been examined, it is doubtful whether we are justified in giving the disease a strictly anatomical name.

Although, as Charcot contends, the stress of the disease probably tells on the motor cells of one or other anterior horn, and it may be that the other changes found in the cord are secondary, yet there are clinical reasons suggesting that in some cases at the onset there is a much more extensive disturbance of grey matter, perhaps even cerebral as well as spinal. Moreover, can any more conspicuous example be given of the failure of the anatomical characters when taken alone as the basis of nosology than the similarity of lesion found in two such different diseases as infantile paralysis and progressive muscular atrophy? Vulpian's suggestion to get over the difficulty by the assumption that one is an acute, the other a chronic form of the same disease, is surely special pleading.

As stated by the author, in typical infantile paralysis, considered clinically, the morbid action is definitive in its distribution in the sense of not extending beyond a certain area attained very soon in the course of any given case. Of the diseased structures included within that area, certain of them may get worse, but others tend to return to the normal condition.

It is on the ground of the second-mentioned characteristic that the author bases his title, *regressive* paralysis.

With respect to the "definitive" area of the disease, it is, however, only fair to remember that there have been some cases where, according to Charcot, "the paralysis in place of attaining at one stroke its highest degree of intensity, has become developed in a progressive manner in the space of a few days, or even a few weeks, and a few others, in which during the period of regression there have been aggressive relapses." I have seen one such case. But these exceptions do not invalidate the general distinction from progressive muscular atrophy where the paralysis follows the atrophy instead of preceding it, and the disease extends from one muscle to another.

The author gives a careful *résumé* of the microscopic researches made especially by the French physicians on this



disease, but as he contributes no new facts, it seems best to consider specially his own observations on the clinical features of regressive paralysis.

This is the first paper, so far as I know, in which attention is drawn to the frequency of onset of this disease in the two hottest months of the year. "Of fifty-three cases in which the date of the attack could be fixed with accuracy, twenty-seven occurred in the months of July and August."

In out-patient practice, the long interval which has generally taken place between the first occurrence of the paralysis and the time when a child is brought under observation, renders it difficult to obtain reliable information as to immediate antecedents; but, judging from the facts elicited by the author, it would appear desirable in future to enquire more closely into the possibility of heat stroke as well as of local cold exposure.

It is very significant that out of sixty-three cases, after careful analysis, the author is only able to associate five with troubles of dentition. It may be safely asserted that of all the "*dei ex machinâ*" which have hindered the study of children's diseases, and which have alike condoned the indolence of the doctor and the carelessness of the parent, none has been more harmful than the indiscriminate reference of infantile diseases to the result of teething.

Without denying the manifold disturbances, nervous and otherwise, associated with the eruption of teeth, we are justified in requiring some careful accounts of the nature of the local irritation in cases where the teeth are held responsible for inducing organic disease in other parts of the body.

The careful investigation made of late years on so-called reflex paraplegias appears to show that they are mostly resolvable either into hysterical conditions or ascending neuritis; and these results would suggest a more rigorous enquiry into all cases of organic nervous disease assumed to be due to reflex action.

With respect to the invasion of the disease, the author very justly lays great stress on the febrile stage which probably exists at the onset for some days, although frequently ignored. It is exactly this period which deserves most study. It is disappointing to find no account of the pyrexia. The only

fact I have to offer on this point is that in one case, three weeks after the onset, the rectal temperature was 104.4. The author records eleven cases in which convulsions preceded the attack, and in several the convulsions were repeated, which would seem to imply that their significance was more than that of an initial rigor. In several of the cases, also, there is a history of the patient lying unconscious for awhile.

As to the important question of the participation of the brain in the disease, about which Volkmann has no doubt at all, there are a few valuable notes, though one could have wished for more details.

In six cases the author records the existence of facial paralysis for a short period. This is very important; and it is to be noted that amongst English observers Dr. West (p. 240) also states that he has seen temporary facial paralysis, with true infantile paralysis. Now although it may be urged that without post-mortem examination it is impossible to assert that these cases belonged to the infantile paralysis type, yet there is a strong argument in favour of the author's view in the fact that in five of them ultimately certain limb-muscles were "picked out" more or less atrophied.

It is quite true that many cerebral lesions occurring in infants are followed by arrested growth of the opposite side of the body; but I am not aware that in such cases individual muscles are "picked out."

Moreover, the great majority of such cerebral cases are characterised by the existence of some spastic condition or inco-ordination of movements, neither of which appears to have been present in the examples recorded.

About Case 11, where right ptosis, right facial paralysis, and complete right hemiplegia occurred, but where ultimately there was response of all the muscles to faradism, I doubt very much whether the author's view can be sustained.

Cases 5 and 29 also seem inconclusive.

In Case 12 there was loss of speech for some weeks. Unless there were other evidences of bulbar disease, an altered vascular condition of the cortex would appear more probable than the author's suggestion of damage to the hypoglossal nucleus, but whatever the cause, it is of interest as bearing on

the cerebral extension of the disease. The author has an important observation on the not unfrequent cases of crossed-limb paralysis. He found one case in which at the onset all four limbs were affected, but eventually regression took place from the two upper and one lower extremity, leaving only the left leg to definitive paralysis and atrophy. Generally he believes "that the crossed form is the result of regression from one or more limbs in cases where three or all four have been affected."

The occasional observation of pain and hyperæsthesia, to which the author refers, is of importance as connecting the infantile with the adult cases where these conditions are notable features at the onset.

The outcome of the author's cases, and his analysis of them, is certainly in favour of the greater generalization of the disease at first than has been often supposed.

Even in the later stages there are not wanting evidences of greater extent of damage than at first might be supposed.

Thus Volkmann specially dwells on the general arrest of development of the whole limb, sometimes even of the whole of one side of the body, in cases where only one or two muscles of the leg are picked out as atrophied.

I have been struck very often in testing electrically, children who were the subjects of infantile paralysis of some standing, with the distinctly subnormal response to both faradism and constant current in limbs other than those where atrophy was noticeable to the naked eye.

With respect to treatment, the author seems to have had excellent results in a few cases. Some of his recoveries have been in cases that were galvanized, &c., for twelve months.

Some more details as to frequency with which galvanism and faradism were applied in the successful cases, and the sort of home treatment used would have been valuable. But the moral of these cases is to "persevere," and this is much more satisfactory than the pessimism and nihilism of those who base their disparagement of galvanism on the results of intermittent treatment for limited periods.

It seems hard to understand how any man can wholly disparage galvanism after but a single observation of the redness

and warmth brought back to a cold wasted limb, even when there be but scant response of the majority of the limb muscles.

The great value of galvanism, as Dr. Poore has pointed out in these cases is, that it is the best external stimulus we have to the nutrition of the atrophied member.

Moreover the power of compensation of both muscular and nerve structures is so great, that even if only a vestige of response be obtained at first, it is fair to hope for some benefit from long-repeated trial, and that "responding" structures may in some degree take on the function of parts which are atrophied.

Friction by the parent at home and attention to warmth are doubtless quite as important as galvanism by the doctor, but most important of all appears to me the lesson taught us by the Swedish gymnasts, viz. of inducing as early as possible nervous influx from the child's own cerebro-spinal centres into the partially paralysed limb.

Where it is impossible to get a child to stand, it has appeared to me that the best results follow from the nurse's frequently performing such movements of the partially paralysed limb as evoke some antagonism, however feeble, in those muscles of the limb which remain unparalysed.

THOMAS BARLOW.

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*Habit and Intelligence.* By J. J. MURPHY. Second Edition. Macmillan and Co. London, 1879.

THE object of the book is "to show the necessity for recognising in all life, both organic and mental, an intelligent agency which cannot be explained as a resultant from unintelligent forces."

This it seeks to accomplish by an examination of the results of recent biological investigation, and thus attempting to ascertain what are the chief laws which these facts exemplify, so as to determine whether or not these laws are derivable from the more general laws which obtain in inorganic nature.

The work is described as "a series of essays on the laws of

Life and Mind," but it has not the fragmentary character that might have been expected; all the parts being mutually dependent, so as to form a definite whole, with a distinct purpose.

It is written in a popular manner, and is to be reckoned among a great number of publications, similar in style and aim, that have appeared since the recent floods of light which have been shed upon these subjects; but this one is above the average, the material being not only of wider range, but better digested than is often the case; moreover, the author neither blindly adheres to, nor furiously opposes, any party doctrines.

The greater part of the work is descriptive, being an exposition of the writings of Darwin, Wallace, Carpenter, and others; but selection is judiciously made from the abundant material existing, comprising Physics, General Biology, Psychology, Sociology and Morals, and even Metaphysics; the light, therefore, of the book is mostly transmitted, but condensed and brought to a focus; and at the same time, though making allowance for the extreme suggestiveness of the source of information, the light receives addition during transmission, the novelty being chiefly in the aspects given to the subjects under discussion; though perhaps there is not much that is absolutely fresh in the way of criticism. Those familiar with the leading works on these subjects may still perhaps be interested; and by those who have not time or inclination for perusal of these works, this will be found an excellent compendium.

Of the first edition, most of the sections on physical science, more commonly known facts and laws, evolution, &c., as well as the more formal parts of science, have been replaced by chapters on some of the more special facts of Biology, and the Psychology has been re-written.

In order to give a somewhat adequate idea of the nature and scope of the work, we will give an outline, with a few extracts, to show its leading features.

In the Introduction, the author states his position, and lays down definitions; he says that "Vital (i.e. belonging to unconscious and conscious life)" principles consist of two

classes—(a) belong to the border land where life comes into contact with inorganic matter and force, such as nutrition and reproduction; (b) distinguished from the preceding, are most important and belong to the inner domain of life itself—the laws of organisation and of mind.” “In this inner domain of life, on which dynamics and chemistry have scarcely any light to throw, we find two principles, which are, as I believe, coextensive with life, and peculiar to it; these are, Habit and Intelligence.” “*Habit* is used in an unusually wide sense: that law in virtue of which all the actions and characters of living beings tend to repeat and perpetuate themselves, not only in the individual but in the offspring. This is a fundamental law of life and mind.”

“*Intelligence*.—It is found in none but living beings;” “it is an attribute of all living beings, and coextensive with life.” But it includes “not only the conscious intelligence of the mind, but also the organising intelligence which adapts the eye for seeing, the ear for hearing, and every other part of the organism for its work. The usual belief is, that the organising intelligence and the mental intelligence are two distinct intelligences. I maintain, on the contrary, that they are not distinct, but are two separate manifestations of the same intelligence, which is coextensive with life, though it is for the most part unconscious, and only becomes fully conscious of itself in the brain of man.” “Habit, in itself, is obviously an unintelligent principle.” “But . . . the question arises whether intelligence is an ultimate fact, incapable of being resolved into any other, or only a resultant from the laws of habit. This is by far the most important of all questions now under scientific discussion, and perhaps the most important that Science can ever have to consider.” “This question divides itself into two: the one concerning the unconscious intelligence that organises the body; the other, concerning the conscious intelligence of mind.” Concerning the origin of species, the author says, “I agree with Mr. Darwin that all species have been derived by descent with modification, probably from one, certainly from a few original germs; and further in attaching great importance to natural selection among spontaneous variation, as part of the agency by which

the modifications have been effected. But I altogether differ from him in that I believe the wondrous facts of organic adaptation cannot have been produced by natural selection, or by any unintelligent agency whatever."

About *Mental Intelligence*, Mr. Murphy says he has "come to a conclusion fundamentally opposed to that of the dominant psychological school in this country, founded by Hartley, and to which Mill, Bain and Spencer belong; . . . the characteristic point of whose theory is to account for the whole mental nature by the single principle of association, or, as I call it, *Mental Habit*. I maintain, on the contrary, that in all mental intelligence, as in all organising intelligence, there is an element not derived from habit, and not resolvable into any unintelligent agency whatever."

Finally, he endeavours "to show how the science of history is capable of being elucidated by the same principles which have thrown so much light on the development of individual organisms, and of organic species."

In several places "we are brought to a region usually regarded as out of the domain of science;" "such subjects as the origin of the universe and of life, the nature of intelligence, and the nature and ground of the moral sense, suggest questions, which, if they are to be answered at all, must be answered from data which are not to be found in the visible world." "On account of their transcendent importance, they are treated of separately in the author's 'Scientific Bases of Faith.'"

The first few chapters deal with the elementary facts of life. Organic chemistry is declared to be absolutely distinct from inorganic; "for it is improbable that albuminoids can ever be formed except by living beings." Here, it seems to us, that the author credits the science with a maturity to which it is not entitled; for it is growing rapidly in a truly positive method, by repeated analysis—just as the fertilised germ divides and becomes a "mulberry mass"; but this stage is as yet far from being reached in organic chemistry, and it is impossible as yet to say to what extent integration may not be carried when form at length shall have appeared in it. Life is said to *guide* physics and chemistry, through which it works.

In Chap. III. there is an outline of general physiology, mostly derived from Carpenter's work. The nutritive system is said to transform matter, energy becoming potential (vital energy); and this again is made kinetic—as heat and motion—by the nervo-muscular system. In a note, alcohol is supposed to act by altering the proportion of heat to motion thus produced; the sum-total remaining the same, more motion is produced at the expense of the heat. Life is thus defined: "An organism consists of a mass of peculiar chemical compounds of high complexity, and contains a peculiar kind of energy. Life consists in the constant transformation by the organism of matter and energy into these peculiar forms, and the equally constant transformation and parting with matter and energy." This definition is unsatisfactory, for it is merely an incomplete description of the vital processes: it omits the essential attribute, viz. persistent identity of the whole, while pervaded by constant change of its parts. He does not discuss other theories of life, nor does he anywhere incline to controversy for controversy's sake. In a note he mentions and rejects Radcliffe's theory of muscular action.

Chap. III. deals with the Origin of Life. The author thinks experiments point against spontaneous generation. He applies the term "vital principle" to the agency that produces organisation, and causes the vital functions to go on, for we are entirely ignorant of its nature. Probably life originated by a definite creative act—a view which is strengthened by the fact of the definite commencement of the present state of things (quoting Clerk Maxwell); but a succession of creative acts is not impossible.

In Chap. IV. life is said to be the cause, not the effect of organisation; and although the possibility of much ultra-microscopic structure is admitted, yet this is considered impossible to account for peculiarities of germinal matter: and the hereditary transmission of functional peculiarities is declared to be incapable of explanation by such hypothesis. We confess that this does not seem so self-evident to us. The hypothesis of Pangenesis is not referred to here. There are said to be three chief relations in physical science—Causation, Resemblance, Purpose: causation predominating in the typical



physical sciences, resemblance in the classificatory, and purpose (the relation of special structure to special function) in biology, where causation is least seen. Organisation is defined as the adaptation of structure to function; and the relation of structure to function is said to be the same as the relation of means to purpose.

In Chap. IV. development is described as the transformation of germinal into formed matter; repair is an example of development. Differentiation and integration are said to be the direction of development. Besides these, the organism presents relations of *dependence* and *subordination*; the former being an extension of Comte's series of the sciences within the domain of biology—animal life depending on vegetable or nutritive life, mental life on animal life. But subordination is seen only in the organic world, life working through physical and chemical properties; conscious functions through unconscious ones.

In Chap. VIII. a table of the organic functions is given, thus—

Unconscious	{	Nutritive or Vegetative.	{	Chemical	{	Forming organs.
				Structural		„ tissues.
Unconscious	{	Motor or Animal.	{	Spontaneous.	{	Reflex (stimulus unfelt).
				Reflex (stimulus unfelt).		Consensual (stimulus felt).
				Consensual (stimulus felt).		Voluntary.
Conscious	{	Sensory.	{	Mental.	{	

Chap. IX. *On Habit and Variation*.—It is said to be an example of Habit that acts tend to become easier on repetition. All and none but vital actions become habitual. All mental and motor acts are habitual, except those that are directed to conscious purpose by voluntary impulse. Habits are changeable. *Laws of Variation*.—Some variations are spontaneous: the possession of one habit facilitates the acquisition of a similar one; habit gains prominence by recent use, tenacity by prolonged duration; thus, after disappearing in one generation, it may crop up in another—reversion. Active habits strengthen, passive (i.e. stimulation not followed by any response on the part of the organism) weaken by repetition. The laws of Habit do not account for every particular habit, for they throw no light on the first of a series of habitual

acts. Slight external changes are beneficial, great ones injurious; therefore great variations can only be brought about gradually. Adaptation is admitted but unexplained.

Chap. X.—Though believing in Evolution, Mr. Murphy considers a guiding intelligence essential.

Chap. XI.—*The Facts of Variation*—is mainly a summary of Darwin's 'Variation of Animals and Plants.'

In Chap. XII. the sufficiency of natural selection is considered, and found wanting. Tait's argument of the small chance a single case of favourable variation would have is quoted; also the less endurance and prolificness of high organisms tells against their race.

Chap. XIII. contains some interesting statements:—that allied species frequently present parallel (i.e. having a close correspondence) varieties; and the same is sometimes seen with regard to genera and species; and thus several species of the same genus may be derived from different genera. This is corroborated by the specific characters sometimes appearing before the generic in an individual. Sometimes a series of species in one genus correspond thus with a series in another. This argument has been brought forward by Prof. Cope against natural selection, and in favour of innate tendencies of development: and Mr. Murphy seems disposed to concur; but surely, among all kinds of spontaneous variations, a similar one would appear in more than one kind of organism, and if favourable would tend to be preserved by natural selection; and the facts of analogous organs would rather support such an application of natural selection.

In Chap. XIV. it is argued against natural selection that the least important characters often become most readily fixed; and certainly such examples show that natural selection is by no means the only means of fixing an alteration. Delbœuf's mathematical law is quoted—that if a few individuals marked by a harmless peculiarity are produced in each generation, their proportion will tend to increase till their number equals that of the representatives of the original stock.

Chap. XV. is on the effect of Change of Conditions. It produces a tendency to general variability; very often a certain change of habitat, temperature, &c., will produce a

definite change in the form of the organism; well seen in Fungi.

Chap. XVI. contains an account of Mimicry, Colour and Sexual Selection, being largely derived from Wallace's essay ('Macmillan's Magazine,' Sept. and Oct. 1877). Natural selection fails to account for protective mimicry, because of the uselessness of early imperfect imitations; but Mr. Murphy considers the similarity of two kinds of organism is due to the similar effects of locality on each, and that thus the protection is secondary. But we think this would not account for the similarity (e.g.) of insects to leaves or sticks. Mr. Murphy says that it seems undecided whether seasonal alterations are due to protection or to impaired nutrition in winter. The power of imitating is probably primary in animals—e.g. chrysalides match the colour of the box containing them. Colours of plants are probably to attract insects. Sexual Selection is next considered, but regarded as doubtful, for ornamental characters are too constant. Probably (as in beautifully-coloured sea-shells) nature sometimes acts purely for ornament.

Chap. XVII. is devoted to an examination of the evidence afforded by Metamorphosis and Metagenesis. Metamorphosis is defined as "development with change of plan." Examples are given, showing how the occurrence of the changes may be influenced by external conditions, and how they may sometimes be completely lost, there being always a tendency to skip those stages; and the Batrachia are good examples where this tendency may be successful or the reverse. In the Batrachia, they probably seem to be due to adaptation; but this can scarcely have been the case in the higher Crustacea. Most are progressive, though some are retrograde. The Nauplius is the tadpole of Crustacea. Metamorphoses afford evidence in favour of evolution, but against natural selection. In considering the metamorphoses of insects, Sir J. Lubbock's views are given: the change of mouth-type in many is acknowledged to be hard to explain. The maggot form of larva is suggested to be the copy of an ancestral worm. A suggestion is made that the Myriapoda may have been descended from Vermes, and have come to resemble insects by parallel variation.

The metamorphoses of Echinodermata are pronounced inexplicable by natural selection, for they do not seem to be adaptive. The process of Metagenesis is likened to what would be the case if flowers were to be detached before maturing their seed. The Medusa-structure is considered to be non-adaptive, and therefore to be inexplicable by natural selection, for the form would be useless until the Medusa became quite detached.

Chap. XVIII.—*On Structure in Anticipation of Function*—is of importance to the main argument of the book. Self-adaptation and natural selection, being incapable of foresight, are declared incompetent to account for all cases of development—e.g. cannot fit organism for future conditions: thus the gradual tendency in the race of Hydrozoa to establish detachment of free Medusæ is considered to prove foresight in order to procure this character when it should be needed. But the argument is allowed to be inconclusive. Similarly the vertebral structure of the Ascidian larva is considered to be the foreshadowing and foundation of a structure whose use is only foreseen. But this argument seems to us quite inconclusive, because the Ascidians have probably retrograded, and the vertebral structure might have been useful to its earliest possessors. The swim-bladder of the fish is adduced as evidence of preparation for the future wants of the race; but it seems to us that there is no evidence of its becoming complex (e.g. in *Ceratodus* and *Lepidosiren*, which are presented as instances of the contrary) except when exposed to conditions when such would be useful. Mr. Murphy next shows that a leg could not have been developed from a fin by natural selection, for the intermediate forms would be useless; but is it not more probable that fin and leg have diverged from a common simple, type of limb? Thus we do not feel convinced by Mr. Murphy's cases; but we admit, that if he could demonstrate a case of what he seeks to show, it would be of extreme importance to his theory.

Chap. XIX. will interest most readers: it treats of the Origin of man. He is believed to have been produced by evolution: but Mr. Murphy says that his brain is perhaps the only conclusive evidence of the inadequacy of natural selection

to account for modifications. Thus Mr. Wallace holds that primitive man has a large surplusage of brain: Mr. Murphy's position is somewhat different, for he thinks that the large size of the brain is due to the acquisition of language at that early period, but that then the language was in advance of the intellectual necessities of its possessors; and therefore could not have been evolved by natural selection; also confirmed by the very slight difference in size between the brain of the savage and that of the intellectually developed civilised man; and by the assertion of moralists that in uneducated men especially there is a large amount of undeveloped brain-power. But is the possession of language really the essential characteristic of man? and, if so, is it beyond his needs in the primitive state? We ourselves do not consider language to occupy such an important position, but to be rather a *proprium* or *accident* of man—just like the facts that he is a cooking or tool-using animal, or a forked radish, or unfeathered biped; and that the most essential quality is the power of reflection—man thereby attaining a clear notion of his personal identity; and that this would be the chief cause of his subsequent immense intellectual and moral progress. But until anything like a science of Comparative Psychology—or even a complete acquaintance with human Psychology—exists, it will be impossible to light upon the differentia of man, or even to determine whether or not his powers and needs are duly proportioned. At any rate there is very strong *à priori* evidence against superfluous powers. Man's hairless back is also considered, as pointed out by Mr. Wallace, inexplicable by natural relation.

Chap. XX.—In this chapter, geological time is said to be too short for the known series of changes to have been produced by natural selection only, therefore it must have been supplemented by other agencies;—similar examples are also adduced of the apparently rapid evolution of a species. It is also declared that the wing of the bat, &c., could not be evolved through natural selection or self-adaptation: but we cannot feel convinced by such examples; for it seems to us probable that the membranous expansion might have been produced by continual stretching of the integument (natural

selection also operating) just as the mouth-structures of the snake have become distensible: and it is not necessary that actual flight should have been performed at first.

Chap. XXI.—Some minor objections to Darwin's theory are brought forward: and then the tendency to similarity of homologous parts and similar tissues is advanced as sufficient to account for many modifications of growth; thus the necessity of an organising intelligence is shown, though this need not act constantly and uniformly.

Chap. XXII. *Intelligence*.—Formative, motor, and mental functions are all guided by intelligence, though this need not be conscious. There is gradation between unconscious and conscious action.

Chap. XXIII. *Instinct*.—Special instincts are as difficult to account for as are special organs. The movements of *Spermatozoa* are similar to food-seeking Instinct. Mr. Murphy considers it impossible that this or the complex instincts of the frog can have been formed by gradual adaptation and inheritance; therefore a self-guiding intelligence is proved. The possibility will probably not appear so inconceivable to all evolutionists.

We come now to the phenomena of Mind.

Chaps. XXIV.–XXVI. deal with elementary facts:—Mind starts from sensation, but sensation alone does not constitute mind. Formation and association of ideas is due to habit, for memory (i.e. permanence) is implied.

Chap. XXVII.—*Time, Space, and Causation regarded as forms of Thought*—begins with an imaginary future criticism on modern metaphysics. Our notion of Time is obtained along with that of permanence of self. The idea of Space is also primitive—obtained with the idea of an external world opposed to self. Mr. Murphy considers that muscular motion is requisite to give us the idea of the third dimension of space; but we think that this might be obtained from sight alone—though this would of course be less complete. Causation is said to be derived from the Mind's knowledge of its own activity; external observation showing merely "unconditional antecedence," and nothing in substance but permanence. But "action implies an agent." "Substance really means an

agent." Here we must express our dissent from Mr. Murphy : for we think external observation gives us the idea of force or efficiency—which is just the property that he considers—and rightly—the essence of Causality—as well as unconditional sequence ; and this efficiency we attribute to the agent—Substance. This is well put forth by G. H. Lewes ('Problems of Life and Mind,' vol. ii.). Therefore it is unnecessary to seek an explanation in pure subjectivity.

Chap. XXVIII. *The Grounds of the Moral Nature*.—Its root is in the sense of pleasure and pain : these serve to guide the intelligence of a sentient organism. Morality consists in three things—prudence, unselfishness, holiness (preference of high aims to low). As in all life, the higher is developed out of, and presupposes, the lower. The author disagrees with Mill's explanation of higher qualities by association.

Chap. XXIX.—Sensation is the germ of mind. Language is essential to thought. Prudence is due to association. Sympathetic feelings are founded on our own pleasures and pains.

Chap. XXX.—A child learns to use the pronoun "I" without habit or imitation.

Chap. XXXI. *Physiology of Mind*.—There is mental nature only in Vertebrata and the higher Arthropoda, where the nervous system is centralised ; and no sensation in the simplest and lowest nervous system. Adopts Carpenter's view of the functions of the parts of the brain. He quotes Prof. Young, of Dartmouth Coll., U.S., that objects illuminated by an electric spark may be seen a second, third, or fourth, time, which he attributes to return of the nervous current to the retina, and reflexion again to the sensorium. Attention is ascribed to concentration of the nervous current in the sensory ganglia, the normal consensual motor impulse being inhibited by a downward discharge from the cortex. In a note, Ferrier's experiments are mentioned, and the movements produced are considered to be due to stimulation of the corpus striatum.

Chap. XXXII. *Automatism*.—An Automaton is defined as "something which can act only as it is acted on." Primary automatism is due to vital intelligence : secondary automatism

is unconscious habit, resulting from education. We do not agree with Mr. Murphy in thinking that the impulse to self-preservation is other than the avoidance of pain. Is consciousness a link in the chain of causation? To ascertain this, Mr. Murphy proposes the further question—How is healthy action accompanied by pleasure; and how do hope and fear affect actions? saying that, “if Automatism were true, health might be painful,” &c. Perhaps it might, but such conditions do not exist, except as an occasional exception. We only know that, as an ultimate fact, benefits are pleasant, and pleasures prompt us to obtain them: we cannot explain them further, so Mr. Murphy’s argument has not very much weight.

He next says, “The Will does not create, but directs energy. . . . If the theory of Automatism is true, the Will is only the general resultant of all the mental forces. . . . It sounds like a merely identical proposition to say that in every conflict of motives the strongest impulse is that which prevails. Yet every one who has performed any act of true self-control or self-denial, knows that such is not a substantially true account of the case, that it is possible for Will, directed by Conscience or a sense of Duty, to prevail over mere impulse.” Now, with regard to the first statement that “will directs energy,” we confess that we do not see how this is possible: for, though it is stated in a note (quoted from ‘N. Brit. Rev.,’ 1868) that “a force acting at right angles to the direction in which a body is moving does no work, although it may continually and continuously alter the direction in which the body moves,” it is not at all clear to us how such could take place without creation of force or disregard to the second law of motion. However, the truth will be plainer to our physically educated readers. A quotation is also made from the ‘Contemp. Rev.,’ June 1878, where it is attempted “to show that a motor may at certain points (points of bifurcation) indifferently take one or other of two different directions—so that an extra physical action might be the effect of a directing power.” However, this is not generally accepted among mathematicians, and we are not mathematical enough to be convinced by it. With regard to the second part of our author’s statement, we think that the



highest motive preponderates because it is most skilfully directed by reason—which outweighs mere brute force of impulse.

Chap. XXXIII. *Habit and Variation in History*.—The Sciences of language, æsthetics and politics, all depend on psychology. In language, habit and intelligence both work—habit supplying the words, and intelligence combining them. Mr. Murphy hopes that in time the science of comparative grammar will be extended to comprise comparative syntax as well as comparative etymology. Language is an organism—constructed by thought to serve as its instrument, as life constructs and uses the body. Like bodily organisms, language presents growth, development, rudimentary organs, variability, morphology. In fine art, the moving power of progress is the love of slight novelty. The same holds in politics; all changes must be gradual; progress consists mainly in the formation of habits. Custom is older than legislation, i.e. unconscious before conscious.

In Chap. XXXIV. the operation of natural selection in history is exemplified. Mr. Murphy criticises Mr. McLennan's view of primitive marriages; though agreeing with him that marriage arose from wife-stealing, he differs in holding that, instead of this being prompted by female infanticide, it was rather due to the desire for domestic life, and the favourable effects of natural selection.

In Chap. XXXV. the individual and social organism are compared.

Having now sketched the contents of the work, we will just glance at its leading features apart from the detail in which they are founded.

His first and main point is to establish the existence of an intelligent principle, i.e. skilful direction towards a purpose, as opposed to blindly acting force.

After this (its existence being proved) comes the inquiry as to its nature. Mr. Murphy considers, and endeavours to show, that it is something distinct from ordinary matter—that matter *per se* is not invested with properties that might under some peculiarly favourable combinations present the pheno-

mena we know as life. This he proves by an investigation of natural selection and other inorganic agencies, and finding them insufficient to produce the effects that might be attributed to them: and thus, this purposiveness is something superimposed on ordinary matter—which does not, according to him, show “the promise and potency of every form and quality of life” (Tyndall). But his position here is not altogether clear, for it is not obvious whether or not he would ascribe all changes in the inorganic world to the inherent properties of inorganic matter, or whether the most complex, at least, of such processes depend instead on the guidance of those energies by a superior skill.

We have before stated that we think his argument of the power of an immaterial principle to act on matter is inconclusive.

It is not altogether easy to ascertain what meaning he would attach to *Habit*. Whether it is due to the properties of matter, as a resultant from some peculiar conformation, or whether it is an immaterial principle, similar in kind, but inferior in degree of skill to intelligence; probably the former is meant, for it is not denied to be derivable from an unintelligent agency. But he ascribes to habit the tendency to become easier on repetition; and this to us seems to be rather the *effect* of habit, and to imply intelligence; and thus his distinction between habit and intelligence does not seem to us to be absolute, but that both are examples of intelligence; in the one case acting as adaptation, in the other as reproduction. Certainly, he says that intelligence is exercised in every formative and motor act; and perhaps he means that in every habitual act, it is intelligence acting *through* habit, as life works through physics and chemistry.

That natural selection fails to give a complete explanation of organic variation, or even to be the chief agent, we think he amply proves; in this, his arguments much resemble those of others, especially Mivart; in fact, his main weapon—structure in anticipation of function—is almost identical with Mr. Mivart’s, of the uselessness of incipient structures; but, as we have before said, we do not think the arguments unanswerable, for it is impossible to say what may or may not

have been the use of an organ in that stage, nor even what that stage was like, for its early and subsequent uses may be widely different. Mr. Darwin's supposition concerning the baleen of the whale is a case in point. Mr. Murphy seems to argue as if we ought at once to be able to predicate the use of every particular structure; but surely this is assuming for ourselves a power of penetration to which we have no claim; for though analogy justifies us in assuming that every part probably is of some use—great or small—to the organism, yet it may be long before we learn what that function is. But Mr. Murphy acknowledges the frailty of the evidence in support of such preconceived and designed adaptations, except as applied to the brain of man, a case which we consider perhaps less proved than the rest. To us it seems far simpler (a justification Mr. Murphy perhaps would not allow, although we merely use it hypothetically) to assign such harmony of function to need to a power of adaptation, inherent in the organism by virtue of its constitution, and of whose life it is the ultimate law, special adaptations and habits being but examples of it: Mr. Murphy looks on it in the opposite way, considering habit and intelligence ultimate, and adaptation but special cases of these. With respect to the question whether an organism has an innate tendency to develop, Mr. Murphy evidently answers in the affirmative, though he ascribes it to intelligence, working by means of the simple qualities of the organism; and in this respect he appears to differ somewhat from Mr. Mivart, who lays great stress on this innate tendency as the chief agency of progress, comparing (after Mr. Galton) it to a multi-faceted spheroid constantly seeking a more stable equilibrium; while Mr. Murphy dwells as strongly on the intelligent adaptation to outward circumstances, though recognising the inherent tendency to progress; thus also he differs from Mr. Spencer, who denies any inherent tendency to progress. In this, we are disposed nearly to coincide with Mr. Murphy, for we think that a constant tendency to a more stable internal equilibrium is as strong as the constant tendency to equilibrate internal with external conditions.

Concerning the nature of the intelligent principle, Mr.

Murphy entirely discards the belief that the skilful direction of the organism is resolvable into the properties of its constituent parts; and he does not seem to think that physics and chemistry will reveal to us properties of matter more subtle than we at present know it to have. In this we think he is premature; for though we are by no means *certain* that matter can evolve life; yet, in the present imperfect state of the sciences on which biology depends, it is not unreasonable to expect the future demonstration of material attributes superior to what we can as yet suspect, expect from speculation concerning their supposed effects. It is this prematurity of conclusion—the hasty desire for explanation before the strict laws of logic warrant—that we deem the chief fault of the book. It is really akin to the errors of ultra-positivists; the differences being, that whereas they try to fit every phenomenon into accordance with their well-established though too narrow laws, he produces an efficient law by fiction, of the imagination.

His philosophical position therefore differs from that now tending to prevail in being dualistic, not monistic. Were it not so, his position would have close affinity with the idealism of Schelling and Hegel; or the modifications introduced by Schopenhauer and Hartmann; a class of philosophy with which (apparently Hegelianism) he acknowledges, in one or two places, his sympathy (p. 412). For the same reason he, as he takes the trouble to assure us, is not a Pantheist, for he denies consummate power to material substance.

Because he alleges the absolute distinction of intelligence from matter, we cannot identify him with Hartmann, to whose 'Philosophy of the Unconscious' his position otherwise bears the strongest resemblance; in that it expounds how an unconscious intelligence becomes conscious in the course of its evolution; but Hartmann identifies intelligence with matter and energy as the absolute reality. They are especially alike in the view taken of, and position assigned to, the Darwinian theory. An important difference of Murphy from Hartmann is the power of distant foresight which he supposes the intelligence occasionally to exercise: it is this latter opinion that gives Mr. Murphy such a peculiar position as compared with

the philosophical systems referred to; and it also brings him into close relation with the formerly dominant teleology of Paley and others. His proof of this clairvoyance seems to us sadly lacking in weight; but we admit the soundness of his conclusions if he could establish complete demonstration of such a case. Briefly stated then, his position seems to be a dualistic (designer *versus* realised plan) teleology, modified by concessions to monistic evolution, and with a suppressed inclination to absolute idealism.

Before concluding, we desire to express our great satisfaction with the style of the book; not merely as to the manner of its composition (it being very readable), but also to the spirit in which the argument is conducted. For, though frequently expressing his dissent from currently accepted opinions (both orthodox and heterodox), yet his language is free from bitterness and animosity; and though he often dogmatically announces his own opinions, yet it is done so frankly that he disarms us from fear of lurking prejudice; and it is evident that the author is stimulated in thinking and writing by that purest of virtues, the desire for the acquisition and propagation of truth.

F. L. BENHAM, M.B.

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*The Brain and its Diseases.* Part I. *Syphilis of the Brain and Spinal Cord.* By T. S. DOWSE, M.D. London: Baillière and Co., 1879.

THE remarkable advance of pathology is to be seen in many ways, but in none more than in the direction of work in the great asylums—asylums for the insane, the aged and the incurable. Twenty years ago it was sufficient to have changed lunatic asylums from dungeons into hospitals: the reform of workhouse infirmaries dates from a still more recent time, and the proper care of incurables has yet to be undertaken. The administrative reform of such retreats is then barely accomplished, and the use of them for scientific observations must follow these more obvious reforms. Even at present, although lunatic asylums have been first in administrative reform, yet

in by no means all of them, perhaps not in many of them, is scientific study adequately encouraged. The names of a few asylums which have rapidly advanced in this direction occur at once to the readers of 'BRAIN;' but we fear the very distinction of these presents some of the brilliancy of contrast. The wealth of pathological material lying unheeded in our workhouse and incurable wards is enormous; and Dr. Stretch Dowse is already favourably known as one of the first of the profession to "exploit" the riches of such hospitals as the Central London Sick Asylum at Highgate. It is interesting to add that in his preface Dr. Dowse acknowledges a debt to the nurses of that hospital, "without whose vigilance and kind co-operation many of the minute clinical records would not have been fully brought under observation." Such an acknowledgment as this seems peculiarly honourable to both parties, and to mark as great a reform in the quality and self-respect of the nurses as in the attitude of the resident physician no longer possessed with the desire to keep those nurses 'in their places.' The invention of the clinical thermometer alone has done more than any one other thing to elevate the nurse and to bring her work into harmony with that of her medical directors. Dr. Dowse may therefore be said to have had one of the first chances in England of developing the resources of a sick asylum, after the example of Dr. Charcot at the Salpêtrière; and, without the presence of the volume now in our hands, he has shown already that he was not only alive to his opportunity but capable of taking advantage of it. It would appear from the title-page that Dr. Dowse intends in this and succeeding parts to complete a survey of the "Brain and its Diseases"—an arduous task, in which we wish him success and a long life. The author has begun his task by publishing an essay "On Syphilitic Disease of the Nervous System," and this essay is the subject of our present article. Its contents are: The history and nature of syphilis; the diagnosis of syphilis of the brain and spinal cord; the diagnosis of syphilis of the sympathetic system and of the peripheral nerves. Then follow chapters on the treatment of syphilis, on hereditary syphilis, and on syphilitic epilepsy. The chapter on Pathology closes the work, an arrangement which seems to

have commended itself more strongly to the author than it is likely to do to his readers. The chapter on the nature, or, as it would be better entitled, on the features of syphilis, contains but little, and that little does not profess to be novel. Yet what is said has the force of the master of legions, for Dr. Dowse speaks with ten thousand patients at his back (p. 4). Hence dicta of the following kind have from him a weight which they would lack in the mouths of less fortunate observers—namely, that hard and soft sores alike may be followed by constitutional symptoms, among which may also come the hardening of the seat of the forgotten or half-forgotten primary abrasions; that shocks physical and mental may determine the appearance of a syphilis dormant for years previously; that among the several indications which betray a syphilitic habit a thickening of the rectum takes as high or a higher place than choroidal patches, faint keratitis, lingual psoriasis, and the like; that active syphilis does not ally itself with gouty, rheumatic, tubercular, scrofulous, or even cancerous diatheses, but rather suppresses them; that syphilis of the nervous system (excluding the osseous system), and of the thoracic and abdominal viscera seem to stand almost inversely to each other; that under adverse circumstances changes from period to period are so rapid in syphilis and so indefinite as to bewilder the observer trained in the stricter schools; and so forth. Such points as these are given forcibly and simply, and only so far as may be needed for the purposes of the book itself. Under diagnosis of syphilis the ophthalmoscopic signs are incompletely described, and scarcely advance, if at all, upon the records of ten years ago; and we should have been glad to know more of Dr. Dowse's experience of percussion of the skull. In the chapter on Diagnosis the reader will probably turn aside from the well-known forms of cerebral syphilis affecting the Sylvian regions, the orbital nerves, and so forth, and will turn to see what is said of the forms more recently discussed and more anxiously watched—what is to be made out of syphilis of the cortical regions and of the pia mater; what of the distinction between syphilis of the frontal and fronto-parietal convolutions as opposed to general paralysis of the insane, and to sclerotic degenerations in and

about the medulla oblongata; and what of the far-reaching disease of the middle and inner coats of the smaller arteries, lately described by Heubner, and in 1868 by the present writer in 'St. George's Hospital Reports.' Dr. Dowse does not fail us in these inquiries; and we may confidently refer our readers to these sections of his volume, which are full of instruction, and, like the whole of the book, are based upon an excellent selection of recorded cases. Many of Dr. Dowse's recorded cases will have indeed a classical value, however far the comments upon them may be superseded by his own later work and by later authors. Dr. Dowse goes so far as to say (p. 26) that "certain diffuse changes, revealing psychical derangements, are rarely associated with other than syphilitic growths when combined with objective signs of paralysis." I think most physicians will admit that such cases have hitherto been to them food for conjecture rather than of certainty. On page 32 Dr. Dowse states that "hyperplastic inflammation of the pia mater of the cord is not so uncommon in the second stage of syphilis," and cases are given; and he denies that paraplegia, if syphilitic, is a late event in any case. On the contrary, he says, "it is rare to find a true paraplegia unassociated with brain disease, and seven years after the primary manifestations" (p. 35). This, on reflection, I certainly think is borne out by my own experience, though I had not arrived at such a generalisation. On the other hand, I would urge that myelitis occurring in the course of syphilis as a rule seems to fail in showing the meningeal and vascular changes we look for in syphilis.

In a paper in the third volume of 'The West Riding Asylum Reports,' on "The obscurer Neuroses of Syphilis," the reviewer ventured to attribute to syphilis certain disorders of the temper, of the spirits, of the sympathies, and the like, as well as sleeplessness, simple epilepsy and other simple neuroses, so that he finds a special interest in Dr. Dowse's chapter on hypochondriasis, hysteriform states, sympathetic depression, and so forth, as events in the course of syphilis. Interpretations of this kind are as yet hazardous and difficult, but the facts of their coincidence with syphilis and of their cure by antisymphilitic treatment are too many to be neglected. It is curious that so



little seems known of syphilitic disease in and upon the peripheral nerves; of such disease Dr. Dowse gives some interesting examples. The chapter on treatment betrays the skilled and independent hand of one who is superior to routine in therapeutics, and who knows the limit of his powers; and this chapter cannot be read without profit. In the author's estimation mercury still holds the high place in which it has long been placed by competent physicians; but the reader will be a little surprised to find mercurial baths and inunctions so summarily dismissed, and nothing whatever said of the treatment at Aix-la-Chapelle. The chapter on Pathology abounds with interesting cases, and sets forth our knowledge in this department with decision and clearness. Dr. Dowse is not disposed to regard syphilis as being injurious to the larger arterial trunks. This first instalment of Dr. Dowse's work is then welcome as a solid and able contribution to the literature of cerebral disease, and as a contrast to the flimsy and useless treatises which issue so profusely from the bookshops of the present day. The volume is well got up and the type is good; moreover the pages are cut, which is a piece of finish not to be overlooked. On the other hand, the letterpress is full of small errors, due to careless printing and proof-reading, errors which are scarcely to be excused: for instance, the last line of page 25 has no continuation that can be discovered. The index is equally ill done and incomplete: for instance, no reference to the *spinal cord* finds a place in it at all. These blemishes can easily be remedied in the second edition, which will surely be called for.

T. CLIFFORD ALLBUTT.

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*Hume.* By PROFESSOR HUXLEY. Macmillan & Co. London, 1879.

A SMALL book by a great mind, this might be called the handy-book of metaphysics, at least of so much as is comprehensible by common souls. Professor Huxley, stooping to conquer, excuses himself for the fact that in this little treatise more is seen of his thread than of Hume's beads, on

the ground that he is possessed by "an ineradicable tendency to make things clear." Hume himself is clear and strong, "without o'erflowing full" like the Thames; and Professor Huxley punts us down the great river of thought with a love of truth equal to that which artists have for beauty. And what infinite charms there are in the streams of argument and the reaches of wisdom through which we glide so pleasantly under patient and appreciative and skilful guidance. Passing the pleasing biography of the genial and kindly "subject" to the first chapter on his philosophy, is an attempt made by his critic to depict the object and scope of philosophy, reducing the three aims of philosophy propounded by Kant—What can I know? What ought I to do? For what may I hope?—to the first alone. Science, inquiring what do I know? gives the results of thinking. Philosophy, asking what can I know? investigates the processes of thinking as to with what they are and what they can do.

It is curious how strongly we are bound by the senses in the phraseology of mental facts; thus in the first pages we have "the contents of the mind"—"the psychical phenomena of life." A great, if not an insuperable difficulty in writing of metaphysics is to free ourselves from the terminology of empiricism, so that Huxley writes about a psychical phenomenon as if it were not a monster of speech. Neither is it to him; for further on we find that he means thereby merely a mental event, and that he has proposed the term *psychoses* for what Hume calls perceptions, internal events which no one else can know, but the like of which we attribute to others, because we see in them the like effects.

It may be that Professor Huxley insists somewhat too strongly upon "the close and intimate connection between psychology and physiology," for it is not certain that the philosopher may not deal with the problem of the contents of the mind without much knowledge of the physiology of sensation. The physiology of sensation, so far as it is known or perhaps knowable, stands at the gates of mind, and great philosophers have been and perchance may be but slightly instructed thereupon. Sensation is the basis of mind, but the physiology of sense is quite another thing, and it is con-

ceivable that wholly different organs of those we have might have been evolved for serving the senses. It is true we cannot see without the eyes; but there is no philosophical reason why we should not have been made to see through the nerves of the epigastrium. Physiology stands in the outer court of philosophy, but it does not enter the temple. Neither does it seem quite certain that "the methods of investigation which elucidate the true relations of one set of phenomena (the physiological) will discover those of the other" (the psychological). Rather it would seem that it is not so, for the objects of investigation in the one case lend themselves to repeated and varying observation and verification; the same objects being capable of investigation by many observers, while the subjects of investigation in the other cases never exactly repeat themselves, can only be purposely varied within narrow limits, and can by no means be observed by more than one observer. The fact "that the men who have made the most important additions to philosophy—such as Descartes, Spinoza, and Kant—have been deeply imbued with the spirit of physical science" only proves that great minds are largely omnivorous. It would be just as likely in the future that philosophers should be great politicians as physicists, if the public should ever be brought to the belief that the clearest minds would be likely to make the best laws, and the ablest men the most efficient executive.

Indeed there would seem to be less congruity between physical and mental research than between the latter and any other kind of investigation, unless it be mathematics. And this opinion is perhaps not inconsistent with agreement in the author's tribute of gratitude for the efficient aid which physical science has afforded in fighting the battle against metaphysical and other superstitions.

"Physical science in the last fifty years has brought to the front an inexhaustible supply of heavy artillery of a new pattern, warranted to drive solid bolts of facts through the thickest skulls." Physical science has also routed the horde of ghostly ideas and conceptions of spiritual existence which, with spectral wings, have darkened the fair field of human life. Once all men were materialists, more or less, and even

the framers of the Apostles' Creed made the resurrection of the body a dogma of faith. Then came the spiritualists with their "entangling brambles of superstition." Then came physical science to prove the worth of the body, and that mind was but one of its qualities. But to the investigation of this quality, its genesis, and its modes, which, is metaphysics, the methods of physics are but adapted to the earlier stages of the journey in teaching us the physiology of sense and the localisation of thought. It may be that the molecular movements of the brain which result in thought will hereafter be discovered, but the method whereby it will be discovered will not be metaphysical. True it is that upon this basis of sense must be built the whole superstructure of metaphysics. There is no other foundation, and this being sufficient, the edifices of imagination must vanish, burnt out like a firework, and leaving nothing but the smoke and smell of a useless literature.

Professor Huxley, in his criticism of Hume's definition of "a mind as nothing but a heap or collection of different perceptions," says:—

"With this 'nothing but' he obviously falls into the primal and perennial error of philosophical speculators—dogmatising from negative arguments. He may be right or wrong; but the most he, or anybody else, can prove in favour of his conclusion is, that we know nothing more of the mind than that it is a series of perceptions. Whether there is something in the mind which lies beyond the reach of observation; or whether the perceptions themselves are the products of something which can be observed, and which is not mind; are questions which can in no wise be settled by direct observation."

A weighty passage this in support of the views above taken that the methods of physical science are but partially applicable to the investigation of mental products. Scientifically it is rash to apply the words "nothing but" to anything, even where physics seem nearest to the end of their task. But what kind of positive arguments can be adduced about mind and its composition? First, outside facts which all men can observe, as the fact that when the kidneys do not act the mind is destroyed. Of these many men may practically

observe the same fact, although accurately no two men do see the same fact in exactly the same way. There is always the difference of point of view if not also a difference of sense. Secondly, inside facts, which only the one individual man can observe within when they take place. In the nature of things such facts cannot be compared. The signs of them may be compared, but to what extent these signs agree with the facts themselves must always be a matter of the greatest uncertainty. Observation can get no fulcrum for its lever, no rest for its glass, and physical science with its method is excluded. Even the obscure estimate of the internal events of a man's own mind is precarious and difficult. Hume says—

“Should I endeavour to clear up in the same manner [arranged observation] any doubt in moral philosophy, by placing myself in the same case with that which I consider as evident, this reflection and premeditation would so disturb the operation of my natural principles, as must render it impossible to form any just conclusion from the phenomenon.” The critic however observes that “The manner in which Hume constantly refers to the results of the observations of the contents and processes of his own mind clearly shows that he has here inadvertently overstated the case.” It would be little we should be able to glean by observing other “men's behaviour in company, in affairs, and in their pleasures” if we were unable to interpret them by our own internal experience, and when it comes to comparing internal experiences, to fix if possible some standard of interpretation, it is found that no experiences in two people are quite alike, and no methods of expressing them reliable.

Hume applies this general name of “perceptions” to all states of consciousness, thereby differing from Descartes, who calls them “thoughts,” and from Locke and Berkeley, who call them “ideas.” By the latter term Hume designates the reproduction of mental impressions in memory. The term “perceptions” for all kinds of consciousnesses would seem convenient and correct could men agree thereunto, and it would be an immense boon to much-enduring readers if philosophers could adhere to some common terminology. Perception then, according to our author, applies not only to conscious-

ness of senses, impressions, but to that of their relation to each other. Professor Huxley somewhat alters Hume's arrangement of the elementary states of consciousness as follow:—

- A. IMPRESSIONS: A. Sensations of the five senses, and of Resistance [the muscular sense]. B. Pleasure and pain.  
 c. Relations of co-existence—of succession—of similarity and dissimilarity.
- B. IDEAS: Copies or reproductions in memory, of the foregoing.

“Neither simple sensation nor simple emotion constitutes knowledge, but when impressions of relation are added to these impressions, or these ideas, knowledge arises; and all knowledge is the knowledge of likenesses and unlikenesses, co-existences and successions.”

But Professor Huxley points out that the impression men call a relation has no better claim to the title of knowledge than that which we call a sensation or an emotion; that there is only a verbal difference between having a sensation and knowing one has it.

“The ‘pure metaphysicians’ make great capital out of the ambiguity. For, starting with the assumption that all knowledge is the perception of relations, and finding themselves, like mere common-sense folks, very much disposed to call sensation knowledge, they at once gratify that disposition and save their consistency by declaring that even the simplest act of sensation contains two terms and a relation—the sensitive subject, the sensigenous object, and that masterful entity the Ego. From which great triad, as from a Gnostic Trinity, emanates an endless procession of other logical shadows, and all the *Fata Morgana* of philosophical dreamland.”

The chapter on “The Origin of Impressions” will be particularly interesting to the readers of ‘BRAIN.’ The subject is delightfully placed before the reader in well-chosen quotations from Hume, interlarded with Huxley paragraphs full of guidance and suggestion. There are two lines of investigation. 1st. The origin of impressions. 2nd. The steps by which they become metamorphosed into compound states of

consciousness. It might, by the bye, be a question whether they are metamorphosed or developed, whether in fact the impression does not remain as the ground of the thought.

On the first line "Descartes demonstrated that the immediate antecedents of sensations are changes in the nervous system with which our feelings have no sort of resemblance;" and Hume argues that the sensations of sight and hearing are no more like the causes of them than pain is like the steel which inflicts a wound. And he "fully adopted the conclusion to which all psychological physiology tends, that the origin of the elements of consciousness, no less than that of all its other states, is to be sought in bodily changes, the seat of which can only be placed in the brain." Perhaps Professor Huxley would not have done injustice to his convictions if he had used a more forcible word than "tends" in the above sentence.

Hume's argument against a mere casual connection between the modes of motions of the cerebral substance and states of consciousness is a delicious piece of ratiocination, which want of space alone forbids us to quote at length. The gist of it is that "we shall never discover a reason why any object may or may not be the cause of any other, however great or however little the resemblance may be between them,"—though there appear no manner of connection betwixt motion and thought, the case is the same with all other causes and effects. There is no more apparent connection *à priori* between the position of bodies causing motion than that between motion causing thought. "It is only by our experience of these constant conjunctions we can arrive at any knowledge of causes and effects"—"*à priori* anything may produce anything." "You reason too hastily when, from the mere consideration of the ideas, you conclude that it is impossible motion can ever produce thought, or a different position of parts give rise to a different passion or reflection."

In other words, the argument is this. You say that motion cannot cause thought because you are unable to see in what manner it can cause it. But we do not and seemingly can never know in what manner any one thing causes any other thing. All we do know is the succession of events. This we observe to be constant, and we come thereby to call it cause

and effect. We find this succession of events between cerebral motion and states of mind, and we have the same reason, good or bad, for looking upon this constant sequence as cause and effect as we have in all other sequences we are able to observe. All consciousnesses being preceded by cerebral change, and all material changes being modes of motion, we are compelled to believe that mind is occasioned by the motion of cerebral matter.

We have sorrowfully to confess that we cannot understand Professor Huxley's last paragraph in page 81, in which he admits that there may be a real something which is the cause of all our impressions, that sensations may be symbols of that something, "the nervous system an apparatus for supplying us with a root of algebra of fact, based on those symbols—a brain a machinery by which the material universe becomes conscious of itself." Whatever this may really mean, if it is not banter, the admission will supply a parry to the keen thrusts he elsewhere aims at the pure metaphysicians. Whatever it may mean it is not idealism, for he says truly that "the idealist confines himself within the limits of positive knowledge;" and we have no positive knowledge beyond our sensations, and the ideas they originate. We have no such knowledge of the real something, the nervous algebra and the universe conscious of itself.

The view presented to us of *Innate ideas*, both by Huxley and his masters Hume and Descartes, scarcely covers the well-trodden field of that brain-splitting controversy, which so exercised the mind of Locke. Hume thinks innate ideas must either mean ideas which are natural, in the sense of being in opposition to what is uncommon, artificial, or miraculous; but all ideas are of this kind. Or they must mean ideas contemporary with our birth, in which case "the dispute becomes frivolous; nor is it worth while to inquire at what time thinking begins, whether before, at, or after our birth." Descartes defined innate ideas to be certain ideas which arose not from external objects, or the determination of my will, "but only from my faculty of thinking," "in the same sense as we say that generosity is innate in certain families, or that certain maladies, such as gout, and gravel, are innate in others, because children are born with the disposition or faculty of contracting them."



“They are products,” says Huxley, “of the inherent properties of the thinking organ, in which they lie potentially, before they are called into existence by these appropriate causes.” All of which seems very much to resemble the reply which a man might make to one who denied the existence of ghosts. “Oh yes, there are ghosts,” Professor Huxley might say, “for naughty boys make them out of sheets and hollow turnips, and Professor Pepper otherwise manufactures them with greater ingenuity.” But the innate ideas which Locke contended against were veritable beliefs in mischievous errors. They were beliefs in “primary notions,” “constant impressions, which the souls of men receive in their first beings, and which they bring into the world with them, as necessarily and really as they do any of their inherent faculties.” In this sentence Locke sharply contrasts them with the inherent faculties of Descartes and Huxley, with which therefore he could not have considered them identical. They were supposed to be “truths which can be imprinted on the understanding without being perceived.” To say nothing of Lord Herbert’s curious list of innate principles and their characters, Locke himself says that “the idea of God is one of them, which of all others seems most likely to be innate.” The moral principle is another. Nor are these all, for so recent a writer as Victor Cousin recognised the innate ideas of time and space, and wasted his brain in endeavouring to patch up a truce with them, or to make a kind of composition with their creditors, or credulators. Huxley compares innate ideas to the music which is potential in a penny whistle or an organ, and which is brought out according to the organism of the instrument, when the performer, experience, arrives; but the metaphysical notion which has led mankind such a dance is that the mind is a magic flute playing its tunes without a performer.

In his chapter on the “Nomenclature of Mental Operations,” Professor Huxley does himself full justice, commenting and amending the teaching of his master with great power and acumen. “The mental faculties—which are, properly speaking, cerebral functions—are reducible to the groups of Sensation, Correlation and Ideation.”

We take the liberty of abbreviating the author’s sentences,

and of running them one into another, not because the author's well-known composition is not full of verve and compact, but simply because space forbids full quotation, and because we may trust the readers of 'BRAIN' to fill up the interstices.

The irritation of a nerve-fibre pulls, as it were, a bell-wire, and every sensation is the ring of a cerebral particle. If, like a bell, the cerebral ring recorded no after-sign, the only impressions of relation which could arise would be those of co-existence and similarity. The relation of succession implies memory. "But the special peculiarity of the cerebral apparatus is, that any given function which has once been performed is very easily set a-going again, by causes more or less different from those to which it owed its origin. Of the mechanism of this idealism we know nothing at present. The trains of thought which succeed each other in this ideational process are obviously dependent upon physiological conditions, and are influenced by temperament, by hunger, fatigue, stimulants, drugs, &c. The succession of mental states follows the laws of association, by which every idea tends to be followed by some other idea which is associated with the first, or its impressions, by a relation of succession, of contiguity, or of likeness." The author questions Hume's statement that the ideas of memory are more vivid than those of imagination, and that in the latter the mind "is free to transpose and change its ideas." To Hume's ideas, which are Memories and Imaginations, the author makes the important addition of Expectations, in which impressions are projected into the future. The author changes the old names of particular and general ideas into the better ones of *specific* and *generic*, and gives a subtle and effective explanation of how the latter are produced. In a complex idea formed of several impressions some of these agree with the impressions of a corresponding idea; some do not; the former acquire strength by the law of associations, while the latter neutralise each other, as in the formation of compound photographs from many sitters, the points where the faces agree are brought out strongly, and form a *generic* portrait in contradistinction to the specific portrait of any one of them, whose facial peculiarities are left

vague. This effect, however, arises from the force of repetition and not of association, so that the author's simile does not quite go on all-fours; but as he further says that a specific idea is "a mean of the series," the fact comes out that association in this case is repetition. Children have very distinct memories before they can speak, and act upon them in the formation of expectations. Knowing neither the meaning of the words "sugar-plum" nor "sweet," the infant is in full possession of that complex idea which he will afterwards form into the verbal proposition, "a sugar-plum will be sweet." Berkeley's opinion therefore, which Hume endorsed, that general or generic ideas are nothing but particular ideas annexed to a certain term, which makes them recall other ideas similar to them, can only be true of the formation of general ideas, after the use of language has modified the mental development. General ideas of sensible objects may exist independently of language.

The chapter on the "Mental Phenomena of Animals," which will be instructive and new to the public, we may pass briefly. Its teaching is comprised in the following fine sentence.

"As *comparative anatomy* is easily able to show that, physically, man is but the last term of a long series of forms, which lead by slow gradations, from the highest mammal to the almost formless speck of living protoplasm, which lies in the shadowy boundary between animal and vegetable life; so *comparative psychology*, though but a young science, and far short of her elder sister's growth, points to the same conclusion."

The difficult subject of Instinct is broached, but not much drawn upon. Indeed, it is given up as "a curious but apparently insoluble problem, whether they [the instincts of animals] are or are not accompanied by cerebral changes of the same nature as those which give rise to ideas and inferences in ourselves." In man, "hereditary mental tendencies may justly be called instincts, and still more appropriately those special productions which constitute what we call genius."

In his sixth chapter the author skims the cream from several subjects. With regard to Language, he points out the important difference between the trains of mere feelings which form the minds of speechless animals and the trains of ideas of the

signs which represent feelings, which have so potent an influence on the development of the mind of man. From Language to Necessary Truths is but a step, seeing that in a rigid analysis the latter are found to "depend either upon the convention which underlies the possibility of intelligible speech, that terms shall always have the same meaning, so that the necessary truth that  $A = A$  means only that  $A$  shall always be called  $A$ ; or they are propositions the negation of which implies the dissolution of some association in memory or expectation, which is in fact indissoluble; or the denial of some fact of immediate consciousness." He criticises Hume's needless admission of difference between the relations of ideas and of matters of fact as of what are called natural and of geometrical truths. The latter Hume describes as "propositions discoverable by the mere operation of thought, without dependence upon what is anywhere existent in the universe." But, says Huxley, "Suppose that there were no such things as impressions of sight and mind anywhere in the universe, what idea could we have even of a straight line, much less of a triangle, and of the relations between its sides? The fundamental proposition of all Hume's philosophy is that ideas are copied from impressions; and therefore if there were no impressions of straight lines and triangles there could be no ideas of straight lines and triangles. But what we mean by the universe is the sum of our actual and possible impressions."

Superfluous reasons are dangerous things; like unexploded shells lying about an old battle-field they may injure the man who has first used them. Hume has employed some such, which weakens his argument about Cause and Effect. Instead of grounding our belief solely on personal experience and the habit of mental experience thereby produced, he formulates the axiom that "whatever event has a beginning must have a cause," which cannot be proved to be necessarily certain; and he also argues that cause and effect being distinct ideas, and separable in thought, "we may conceive any object to be non-existent one moment and existent the next, without conjoining to it the distinct idea of a cause or productive principle;" which, as the critic observes, is an argument which assumes to every question at issue. In fact, the axiom of causation is, as

Huxley points out, the verbal symbol of a purely automatic state of mind, which would even be illogical if it were not constantly verified by experience; which stores up memories generating expectations in a manner which cerebral physiology may hereafter explain.

The idea of something resident in the cause which produces the effect, and which we call Force or Energy, is an addition to this idea of the necessary connection between the cause and its effect which Hume explains as "the results of the association with inanimate causes of the feelings of endeavour or resistance which we experience, when our bodies give rise to or resist motion." We energise the dead world, transferring the sentiment of a *nisus* to inanimate objects, which is not less absurd than supposing the sensation of warmth to exist in a fire. Even in ourselves we know nothing of the feeling we call power except as effort or resistance, and have no means of knowing whether it has anything to do with the production of motion or thought.

To project this notice further into the interesting and important chapters on theological and moral questions, which conclude the volume, would scarcely be suitable for the pages of this Journal. Our readers must study for themselves (as indeed we trust they will the whole volume) the chapters on Miracles, Theism, Immortality, Liberty, Morals. Suffice it to say that Hume's arguments and opinions are expounded in a masterly manner, and criticised in a just and temperate spirit, and that we have a vivid and faithful rendering of the great sceptic who has slain for us so many "dragons of the prime" that now we dare not only to stand on the marge, but to venture into the ocean of truth, as far as our powers will permit. The great favour with which Professor Huxley's little work has been received is a test of the vast change which has been wrought in men's minds since Hume wrote. To have mended the teaching of his master in many important particulars, trimming and feeding the lamp of philosophy which he carries with loving diligence, is the duty of a true disciple becoming a master to others; and this is what Professor Huxley has done and is doing to great perfection.

JOHN CHARLES BUCKNILL.

## Clinical Cases.

### ON SIMPLE APHASIA, AND APHASIA WITH INCOHERENCE.

BY DR. MAGNAN,

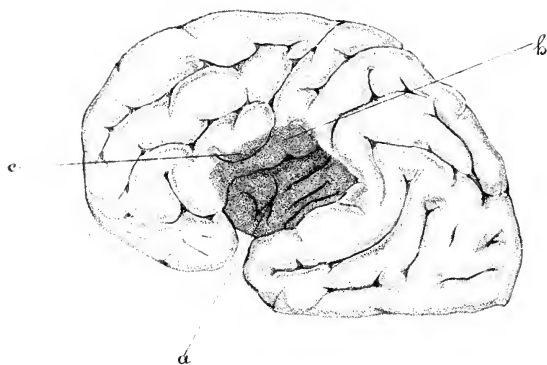
*Médecin de l'Asile Sainte-Anne, Paris.*

THOUGH aphasia, or the loss of speech, commonly presents itself in a simple form, it occasionally, like other cerebral affections, exhibits complications which are deserving of study. Of three cases which I have lately observed, two are ordinary; the third, on the other hand, differs both in its physical and psychical characters. In all three, however, the pathogeny was the same, the primary cause being lesions in the circulatory apparatus. Twice the mitral valve was the seat of atheromatous abscess which gave rise to embolisms of the cerebral arteries; in the third the obstruction originated in the vessels themselves. In all the whole arterial system was the seat of atheromatous degeneration, and infarcts were found elsewhere also, in the kidneys in all and the liver in one case. The cerebral softening was situated in the left hemisphere. The third frontal was the seat of lesion, but with it also, and to a considerable extent, the island of Reil was involved. This is worthy of note all the more as the coincidence of lesion of the insula and third frontal is very common in aphasia, often, indeed, the lesion of the third frontal is slight as compared with that of the island, and in certain cases the insula alone is the seat of lesion.

In the three cases the affection of motility was on the right side, but in one it was limited to the upper extremity.

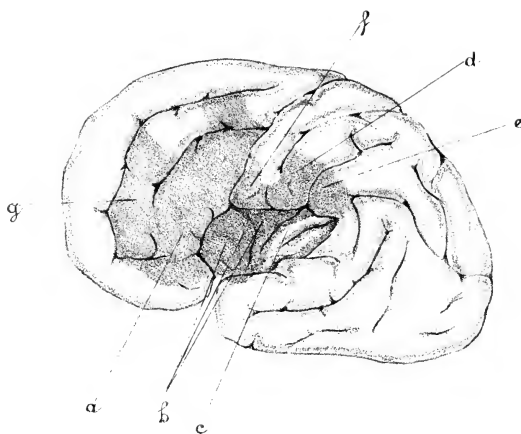


Observation 1.



G. Paul.

Observation 2.



B. Marie. fe



OBSERVATION I., APHASIA.—*Right hemiplegia—Mitral lesion—Plaque jaune of the whole surface of the Insula, and the posterior half of the edge and under-surface of the third left frontal convolution (old lesion)—Softening of the internal capsule (recent lesion).*

G. PAUL, basket-maker, ætat. 40, was admitted into the Asylum on Oct. 25, 1878. No history was obtainable. On being questioned he seemed to understand the majority of the queries, but he could not answer, giving utterance only to the monosyllables "Mo, Ba, Bon," indicating with his left hand that he could not speak.

On being pressed, he became impatient and burst into tears. He looked at, but made not the least effort to read, a book which was placed in his hands, and he could not write. He dragged the right leg, and could with difficulty raise the right arm. A systolic murmur was audible at the apex of the heart, and there were sibilant and sonorous râles in the chest. He seemed oppressed, but there was no fever.

On Nov. 1st he had an apoplectiform attack, with complete resolution of the already enfeebled right side and dropping of the right angle of the mouth. On Nov. 2 there was marked œdema with rise of temperature in the paralysed limbs. Deglutition was difficult, and there was a semi-comatose condition. Death occurred on Nov. 4.

*Autopsy.*—Membranes cedematous. Sanguineous infiltration of the pia mater on a level with the middle of the ascending frontal convolution on left side. Obliteration of the Sylvian artery at its origin, before giving off the branches to the corpus striatum, by an embolus. This was formed in its centre by a cretaceous fragment of the atheromatous abscess of the mitral valve. Round this as a nucleus there was a delicate envelope of fibrin.

Plaque jaune on the whole surface of the insula (*a* Litho.), the foot of the ascending frontal (*b* Litho.), the posterior half of the edge and inferior surface of the third frontal (*c* Litho.). Further, a frontal section showed a focus of softening in the corona radiata, a little above and outside the nucleus caudatus, and above and within the lenticular nucleus. This focus had a length of about 3 centimetres and a breadth of 4 to 5 millimetres. In addition, below this there were two small lacunæ occupying the middle of the internal capsule. The medullary substance between these lacunæ and the focus situated above, apparently normal, was seen on microscopical examination to be markedly altered, and one could discover numerous granular corpuscles, indications of advanced degene-

ration. Further back, in the parietal section, beneath the principal focus, there were lenticular nuclei, also white and indicative of recent lesion. So that the anterior third of the internal capsule was really affected, notwithstanding the apparently normal appearance of the parts between the foci of softening.

The membranes were easily separable from the whole surface of the right hemisphere, and there was no appearance of lesion on the cortex or centrally. The heart was voluminous; its pericardial surface exhibited here and there milky spots; the walls of the left ventricle were hypertrophied. The mitral valve was considerably thickened, and the auriculo-ventricular orifice was narrowed and the valve incompetent. It was surrounded by cretaceous deposits. The right columna carnea, at the point of insertion into the valve, was deeply hollowed out by an atheromatous abscess, at the bottom of which was a considerable quantity of debris ready to be propelled into the aorta. The left column was yellowish and hard at its insertion into the valve, but there were no signs of ulceration. The sigmoid valves were not rigid, and the aorta exhibited a few yellowish spots, slightly prominent but not softened. The kidneys were voluminous. The left exhibited an old infarct of about 25 millimetres in diameter and extending about 7 millimetres in depth. In cutting into the two kidneys the cortical substance was found yellowish, the medullary substance appeared normal. The liver was yellowish and somewhat fatty. It exhibited traces of an embolus.

*Remarks.*—The cardiac affection in this case was the starting-point of the cerebral lesions, but, though having the same origin, they form two groups quite distinct and of different dates. The large cortical lesion described above was the first, and was the cause of the aphasia and incomplete right hemiplegia; the other was the softening of the internal capsule which was the cause of the complete hemiplegia with œdema and rise of temperature on the same side. The seat of the embolus, the origin of the Sylvian artery, explains the extent of the central lesion, and its recent date accounts for the almost normal aspect when looked at only with the naked eye.

**OBSERVATION II., APHASIA.**—*Right hemiplegia—Softening of the third frontal convolution, of the Insula, and other parts of fronto-parietal region of the left hemisphere.*

B. MARIE f<sup>e</sup> D., aged 65 years, was admitted on Dec. 31, 1877, being sent from the hospital, where her cries and excitement during the night disturbed the other patients. B. recognised objects, but she could not call them by name, and

responded to every question by the syllables "ton, ton, ra, ton, ton," accompanying this utterance occasionally with a negative shake of the head. She could neither read nor write. She was affected with right hemiplegia without loss of sensation; she was feverish, feeble, and breathed with difficulty. She died on January 16.

*Autopsy.*—The calvarium was of great thickness, measuring not less than 11 millimetres in the middle of the occipital. The diploe was not injected, and the bone appeared healthy. On incision of the dura mater, 120 grammes of serosity escaped. The arachnoid and pia mater were infiltrated, and here and there opalescent. The right hemisphere weighed 500 grammes, the left 442 grammes. The cerebellum, pons, and medulla weighed 148 grammes. The whole weight of the encephalon was therefore only 1090 grammes. The left hemisphere exhibited a depression in the fronto-parietal region, and through the membranes a yellowish (dead-leaf) tinge was perceptible. The pia mater separated readily from the surface everywhere, except at the softening, where it adhered. The softening occupied the whole of the third frontal (*a* Litho.), the first three digitations of the insula (*b* Litho.), the anterior margin of the fourth (*c* Litho.), while the fifth was normal. Above, from behind forwards, the lower third of the ascending parietal (*d* Litho.) and the neighbouring parts of both parietal lobules (*e* Litho.), the lower extremity of the ascending frontal (*f* Litho.), and to the whole of the second frontal convolution, excepting only about a fifth (*g* Litho.). This vast plaque of softening invaded the whole depth of the cortex, and penetrated across the insula as far as the claustrum.

The membranes separated easily from the right hemisphere, which was normal throughout.

The heart was covered with fat, especially on the right ventricle; and the left cavities were filled with red fibrinous clots, the right with dark clots. The aorta was yellowish, and exhibited numerous spots of atheroma, slightly elevated but not softened. The large arteries at the base were atheromatous; and there were also atheromatous patches on the secondary arteries, in some parts invading the whole wall. The right lung was adherent posteriorly at the lower two-thirds to the costal pleura. The false membranes were red in places, and infiltrated with blood. The base of both lungs was engorged and dense. They could be penetrated with the finger, but did not sink in water. The liver was slightly fatty, and the surface granular after being stripped of its capsule. In the right kidney was an old infarct; the left was of the size of a pigeon's egg, and also marked with small infarcts.

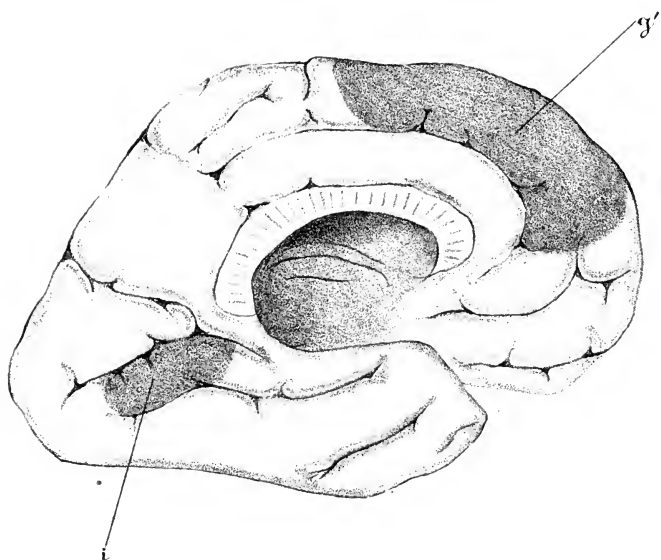
*Remarks.*—The thickness and consistence of the cranium are noteworthy in this case, inasmuch as the patient was already advanced in life, and had atheromatous arteries,—conditions which favour rather thinning of the osseous tissue. If the heart did not exhibit valvular lesions as in the former two cases, yet the arteries were more atheromatous. The softening was of considerable extent, and the whole of the third left frontal convolution was destroyed. But here again almost the whole of the insula was involved. The opto-striate nuclei and the internal capsule were normal. The patient presented the ordinary symptoms of aphasia and agraphia, but, in addition, she had been seized with maniacal excitement, which caused her to be taken to the Asylum. Among aphasics, as among other patients attacked by circumscribed cerebral lesions, it is not rare to see in the course of the malady a development of delirious phases, with either exaltation or depression, sometimes even *délires circonscrits*, which completely alter the physiognomy of the patient, and render special precautions necessary.

OBSERVATION III., APHASIA WITH INCOHERENCE.—*Brachial monoplegia—Mitral disease—Plaque jaune in the margin and inferior aspect of the third left frontal convolution ; the third and fourth digitations of the Insula—Plaque at the upper extremity of the ascending frontal at the base of the first frontal—Numerous plaques in other parts of the anterior region of the hemisphere.*

L. CLÉMENT, *ætat.* 42, bottler, was admitted on Sept. 20, 1878. The patient was found on the bank of the Seine, groaning, and uttering unintelligible sounds. On his arrival he responded to questions with interjections and disconnected words and syllables, a bizarre assemblage of sounds pronounced with a variety of intonation as in ordinary conversation. He appeared to follow a train of ideas, but he had entirely lost the memory of the conventional signs by which they are expressed, and he translated his internal language into an incoherent verbiage. When urged to designate by their names the objects around, he comprehended what was required of him, but he made vain attempts, and gave as every reply, “De beu, de beu, a beubeu, beu beu,” which he uttered sometimes with indifference, sometimes with tears. Otherwise his attitude was singular. He held his right forearm raised and slightly bent, the hand hanging motionless. The fingers when raised fell at once. The voluntary movements of the arm and forearm were possible, but sluggish. The right leg

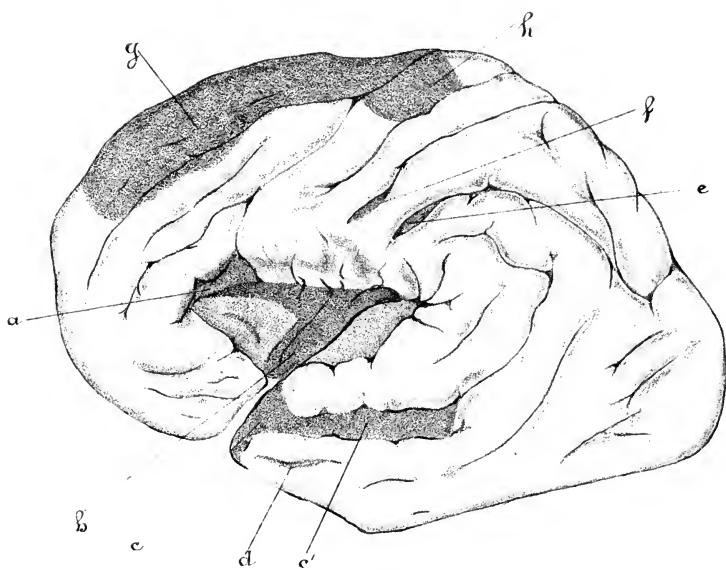


Observation 3.



L. Clément.

Observation 3.



L. Clément

was unaffected and its movements easy. There was no trace of facial paralysis. Sensation was normal on both sides. The patient, feeble and emaciated, seemed to have undergone great privations. On auscultation, a few sibilant râles were audible in both sides of the chest, and the first sound of the heart was accompanied by a prolonged souffle at the apex.

The patient remained till Oct. 18, presenting alternations of calm and excitement, with moans, groaning, and sleeplessness. The aphasia and incoherence continued as before. The brachial monoplegia did not recover, but an apoplectic attack supervened, followed by complete right hemiplegia. The patient rapidly succumbed, and died during the night.

*Autopsy.*—A small quantity of sanguinolent fluid escaped on incision of the dura mater. When this was removed, the left hemisphere was seen to be covered by a thin false membrane, transparent, but injected here and there with fine arborescent vessels. There was no false membrane on the right side. The arachnoid and pia mater were oedematous on both sides, and milky opalescent spots were visible on the middle of the convex aspect of the hemispheres in the region of the Pacchionian corpuscles. The arteries of the base exhibited here and there patches of atheroma. The right hemisphere weighed 575 grammes, while the left weighed only 485 grammes. The temporal lobe of the left hemisphere was closely glued to the third frontal at its anterior and upper extremity, and exhibited a yellowish depressed plaque, which crossed the fissure of Sylvius, and extended into the lower margin of the frontal lobe. These lobes being carefully separated from each other, the island of Reil was exposed, and its lesions rendered visible. The softening, of a dead-leaf tint, involved the lower margin of the posterior third of the third frontal convolution (*a* Litho.), the third and fourth digitations of the insula (*b* Litho.), anterior extremity of the first temporal convolution which was totally destroyed (*c* Litho.), the anterior half of the parallel fissure which has yellowish (*c'* Litho.), but on separation of the two convolutions was of a greyish tint. A small patch of softening was also seen at the bottom of the sulcus which separates the second and third temporal convolutions (*d* Litho.), and another was found at the anterior extremity of the intraparietal fissure (*e* Litho.) and at the inferior extremity of the fissure which bounds the ascending parietal posteriorly (*f* Litho.). In addition, the first frontal convolution was almost entirely destroyed within and without by a patch of softening (*g* Litho.), which, at its middle, slightly invaded the second frontal, and extended backwards as far as the ascending frontal at the point corresponding to the base of the first frontal, and slightly below

(*h* Litho.), but the highest part of the ascending frontal and the paracentral lobule were intact.

Finally, still another patch of softening (*i* Litho.) was found in that portion of the lingual lobule which bounds the cuneus.

The right hemisphere also presented a patch of softening. It occupied the anterior half of the second temporal convolution, and extended inferiorly to the middle of the third temporal, occupying a space of about 2 centimetres. In depth it did not penetrate beyond the grey substance, except slightly about the middle.

The heart was fatty, voluminous, and exhibited concentric hypertrophy of the left ventricle. The mitral valve was completely cretaceous, rigid, and both narrow and incompetent. The margin of the left valve was deeply ulcerated, and there was a large anfractuosity filled with atheromatous debris and small fibrinous clots ready to be detached and launched into the circulation. The sigmoid valves were thin and supple. The internal tunic of the aorta was yellowish, and marked by small patches of atheroma slightly elevated, but not eroded. The right kidney was the seat of an old infarct.

The spleen was small, but normal. The liver was somewhat fatty. The lungs were congested at the base and posteriorly, the right somewhat emphysematous.

*Remarks.*—Though no history was obtainable, yet the fatty degeneration of most of the organs in an individual aged only 42 pointed to alcoholism as the probable cause. From this, and probably also rheumatism, the mitral valve became the origin of emboli which caused the cerebral softenings. Two of these plaques, in the left hemisphere, that which affected the third frontal convolution and that which involved the upper extremity of the ascending frontal convolution, correspond to precise localisations. They sufficiently explain the aphasia and the paralysis of the arm. This double localisation was foreseen, and we expected to find either a vast area of softening extending from the third frontal up the ascending frontal, or two isolated lesions. In these relations numerous clinical facts come every day in corroboration of physiological researches. Does the incoherence depend, as some facts would seem to bear out, on multiplicity of lesions in the cortex? But where should these be situated, and what should be their number and extent? We are as yet unable to answer these questions. We know, however, that they are not to be looked for in the occipital lobes, nor perhaps in the posterior half of the hemispheres. I myself have frequently observed extensive lesions in the occipital lobes without incoherence. In a hypochondriacal melancholic in particular, whose insanity was quite systematic, and had no appearance of incoherence, I



found, on post-mortem examination, two large ochreous patches covering the occipital lobes like a calotte, which extended forwards over the hemispheres as far as the temporal lobes, which were themselves largely involved. In this melancholic the sensibility was impaired but not abolished. It is therefore towards the anterior parts that we must direct our attention, and I do not doubt but that new observations carefully made will throw light on this question of pathogeny.

In the case before us the multiple lesions had transformed the cortex of the frontal and parietal lobes into a veritable mosaic, and the relations between the different parts of the cortex were profoundly altered. What then could be the result of such disorder of the different groups of associating fibres but a state of incoherence? In these facts, as in idiocy, as has been shown by Professor J. de Mierzejewsky, the cerebral lesions isolate, as it were, the different cortical regions; and instead of all being in harmony, and forming a solidarity, we see the strange spectacle of individuals, with special aptitudes perfectly developed, but isolated, and contrasting vividly with their sterility as regards other intellectual powers, appropriately deserving the name of wise idiots.

Was our patient conscious of his language, or believe that he uttered intelligible phrases? It is probable, but how is this contradiction to be explained? On the one hand, he understood what was said; on the other hand, he did not seem to be aware of the incoherence of his own language, when he gave a determinate sense to unintelligible phrases. This apparent contradiction is not uncommon among aphasics. Many of them, in fact, do not understand words, unless on active efforts to excite their attention, when they suddenly concentrate their faculties on one point. Some even succeed in repeating the word pronounced in their hearing, and indicate the object with the hand; but a moment after, when the same object is again presented, they pronounce some syllable or word which has not the slightest connection with it, sometimes recognising their error, at other times, on the contrary, not seeming aware of it.

We may add further that, clinically, we can distinguish two kinds of incoherence, the one which accompanies chronic insanity, especially chronic mania, and which does not show itself till after the lapse of many years; the other, more rapid, sometimes even sudden in its development, is the result of cerebral lesions, most commonly necrobiotic softening, characterised by apoplectiform or epileptiform attacks.

In the first form, the incoherence is the result of a functional disorder of long standing and slow progress, and the incoherence of language is in relation with the incoherence

of ideas; in the second form, the anatomical lesions rudely interrupt the regular train of functions, and so as in L., the incoherence of language is not altogether indicative of incoherence of ideas. It is a question whether in the incoherence of insanity there are also anatomical lesions. According to the results of necroscopic research, the material changes are not very evident, and histological examination discovers only comparatively unimportant changes, such as fatty granular and pigmentary infiltration of the cells, and fatty granular infiltration of the vessels, modifications which, as will be seen, do not differ from those due to senile changes.

*Additional.*—The above remarks had been penned when I had an opportunity of observing a new case of aphasia, which at first did not appear to differ from the common, but which, as regards the autopsy, had certain peculiarities worthy of being recorded.

APHASIA.—*Right hemiplegia—Neuroglia sarcoma of the frontal lobe.*

CATHERINE —, aged 61 years, was admitted on the 18th of October, 1878, accompanied with a certificate from M. Lasègue, as follows: "Dementia, right hemiplegia, aphasia. Absolute inability to attend to her needs." Interrogated on her arrival, she was unable to give any account of herself. She pronounced words or monosyllables without connection, and could not name objects. She reflected, tried, became impatient, wept occasionally, but was unsuccessful; but occasionally she hit on the right name, on which her countenance expanded, and she seemed pleased at her success. She had the irritability, sensibility, and mobility of aphasics generally.

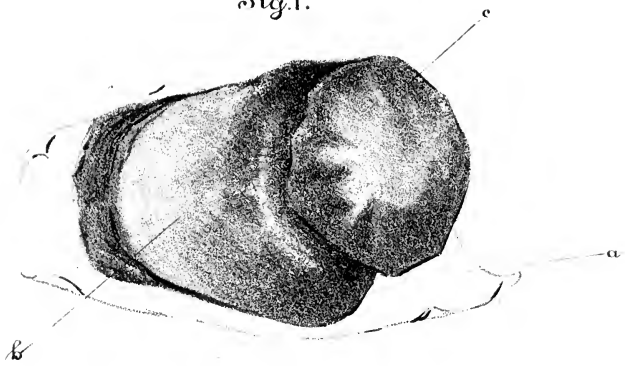
She repeated words pronounced in her hearing, and even occasionally pointed to the object when the name was pronounced in the midst of others. The relation between sight and hearing was preserved. The auditory image revived the visual image (Griesinger, Sander, Jaccoud). On the other hand, if one gave a thing a false name she detected it, and signified the same by a negative shake of the head. Internal speech therefore was retained, but its outward projection was defective.

She did not seem able to read. She held a paper in the left hand (the free one), and looked at the print, but did not follow the lines.

The right hemiplegia was complete; the arm could not execute a single movement, and the right angle of the mouth was slightly depressed. The radial arteries were rigid. There was a slight systolic murmur at the apex of the heart.



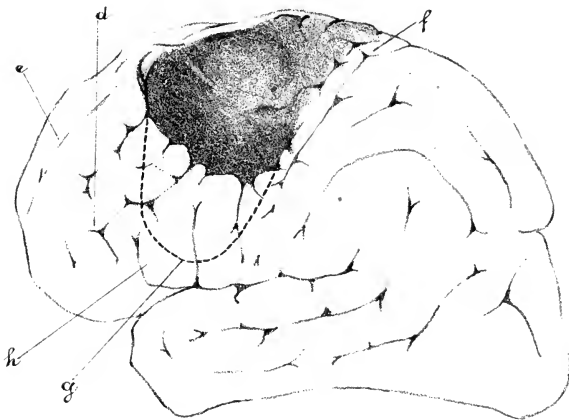
Fig. 1.



*Sarcoma of Neuroglia*

- a. Dura Mater
- b. Inferior lobe where the fibrous tissue predominates.
- c. Superior lobe where the cellular elements predominate.

Fig. 2.



*Indented Cavity in the left hemisphere caused by the Sarcoma of Neuroglia.*

- d. First Frontal Convolution
- e. Second .. ..
- f. Ascending .. ..
- g. Limits of the tumour in the thickness of the left hemisphere.
- h. Third Frontal Convolution.

On Dec. 10 she was found in a state of hebetude at the hour of visit. She did not hold out her hand as was her custom, and did not appear to understand questions put to her. This enfeeblement, due probably to an apoplectiform attack which had escaped notice in the night, diminished during the day, and next day she had regained her habitual attitude. On Dec. 24 erysipelas of the face occurred, followed by a semi-comatose condition, and after various changes for the better and worse, she expired on January 4.

The ensemble of symptoms left no doubt as to the cause of the aphasia and right hemiplegia. The patient having a cardiac affection, it was natural to suppose embolism and softening of the third frontal convolution of the left side, the insula, corpus striatum, and deep parts. On post-mortem examination quite a different affection was actually found.

On incision of the scalp at the level of the temporal fossa of the right side, an abscess in the tissue was discovered, of 5 to 6 centimetres in extent. The calvarium was slightly adherent at the vertex, and presented, anterior to the middle of the frontal, plaques of dull white osteitis. In addition, there was an osseous elevation on the internal surface of the left parietal, on a level with the posterior part of the left frontal region. This prominence, of a bright red colour and mammillated, was raised about 6 millimetres above the internal table over a surface of 3.5 centimetres. It was surrounded by a red zone of 2 to 3 centimetres. It depressed the dura mater, which however remained thin, smooth and polished here as elsewhere. This membrane, incised inferiorly along the external border of the left hemisphere, could be raised from below upwards as far as the upper border of the third frontal. There it adhered feebly to the other membranes. These adhesions, when broken up, which could be readily effected, exposed a tumour inserted in the internal aspect of the dura mater, by a surface of which the diameter was not less than 6.5 centimetres. This tumour, of a conoid shape, directed obliquely from below, outwards and forwards, exhibited at its middle a slight constriction, which gave it a bilobed appearance (F. 1, *b c*). Resting by its base on the inner face of the dura mater, its apex penetrated deeply into the left hemisphere, which it hollowed out and displaced at the posterior half of the first two frontal convolutions, and the upper two-thirds of the ascending frontal. Of this half of the two frontals nothing more was visible than the base of insertion into the ascending frontal, and which was almost entirely effaced. The ascending frontal was deeply depressed and atrophied, principally at its middle third, and the cortex was not more than two millimetres in thickness. The tumour

penetrating the left hemisphere like a wedge, separated and displaced the neighbouring parts, and its apex penetrated as low as the third frontal, and the anterior third of the upper border of the insula. The finger applied to the bottom of the fissure of Sylvius, in the sulcus which separates the third convolution of the anterior border of the insula, found itself separated from the inferior extremity of the tumour by a thickness of cortical and medullary substance, which did not exceed 6 millimetres. The cortex of the posterior extremity of the third frontal, and the upper margin of the first two digitations of the insula, had been, as it were, dissected and isolated by the tumour. It had lost its connections with neighbouring parts, and with the greater part of the fibres of association and transmission. Notwithstanding the immediate proximity of the tumour, the cortex of the third frontal had not been softened.

The cavity of the lateral ventricle was much reduced, and adhesions had formed between the upper and lower walls in the neighbourhood of the sulcus which separates the two surfaces. The digitations of the insula, displaced inwards and outwards, had become very prominent compared with those of the right hemisphere, which were normal. The tumour had some adhesions at the base; was easily enucleable in the rest of its extent. Removed from its cavity, it measured along its oblique margin 7 centimetres, and only 4 on the opposite, which was straight. A perpendicular section showed it to be distinctly bilobular.

The larger lobe in contact with the dura mater was greyish and fibrous-looking, and a fragment examined under the microscope showed that it was composed of a considerable quantity of connective tissue, with fusiform cells. The second lobe was of a vinous red, and presented a small hæmorrhagic focus of the volume of a millet seed. It was mainly composed of polygonal cells of various dimensions, with one or more nuclei, with only traces of connective tissue.

The right hemisphere weighed 625 grammes, the left 600, and the tumour 135 grammes, so that the left hemisphere was far from having lost weight equal to that of the tumour, which had displaced but only slightly destroyed its tissue. On detaching the dura mater from the base of the skull there was visible on the middle of the petrous portion of the temporal bone an orifice of the size of a centimetre, through which it was possible to pass a pair of forceps and extract the auditory ossicles. On the upper surface of the left petrous portion, at its internal third, a second orifice of a similar character was seen. On the two sides the boundaries which limited the loss of substance were excessively thin. The dura mater at this point was neither thickened nor injected, nor were there any traces of

necrosis. Nor were any traces of syphilis visible in other organs. It is therefore probable that the two symmetrical deficiencies on each side of the most prominent parts of the base of the skull were due to a kind of interstitial absorption, caused by the compression of this large tumour, which acted perpendicularly from above downwards.

The heart was voluminous, covered with fat, and there was concentric hypertrophy of the left ventricle. The free edges of the mitral flaps were thickened. The aorta was atheromatous, and exhibited patches slightly elevated but not softened.

The lungs were congested, dense, and friable at the base, and a fragment sank in water. The liver, which weighed 1500 grammes, was free from lesion, and the capsule was smooth and non-adherent. The kidneys were congested throughout, but the capsules were easily separated.

## A CASE OF CHOREA, IN WHICH DEATH WAS CAUSED BY CEREBRAL HÆMORRHAGE.

BY E. BUCHANAN BAXTER, M.D., F.R.C.P.,

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THE extreme rarity of such an accident as hæmorrhage into the substance of the brain in childhood is of itself a sufficient reason for putting the following case on record. But it presents certain other features of importance, some of which may render it an appropriate pendant to the very interesting example of cerebral hæmorrhage in the course of pyæmia narrated by Mr. Herbert Page in the January number of 'BRAIN.'

A. R., a girl eight years of age, was admitted into the Evelina Hospital under my care on January 31st, 1878, with the following history. Her health had been uninterruptedly good, but for an attack of measles, till about three years before her admission. She then suffered from chorea for about six months. The malady cannot have been severe, for she was not confined to bed, and only attended two or three times as an out-patient. No history of fright was obtained, but she was said to have been in the habit of playing with a child who suffered from the same disorder. She had occasionally complained of pains in her legs (termed "rheumatic"), but the joints were never inflamed. There is, however, a distinct history of rheumatism on the mother's side of the family. In October 1877 the chorea returned, and the movements, though never violent enough to take her off her legs, occasionally prevented her from feeding herself. She was subsequently brought to my colleague, Dr. Hayes, who thought she looked too ill for her symptoms, and sent her into the wards at once.

On admission, there was some twitching of both hands and feet. It was quite insignificant in degree, and not more marked on one side than the other. The heart was very care-



fully examined, and its sounds were somewhat unexpectedly found to be *remarkably clear and loud, but unattended by any murmur*. On February 8, the note ran: "often complains of headache. Temperature fluctuates between 98° and 101°, without apparent cause. She can walk without help, and feeds herself. The twitching is very slight." On the 9th she ate her dinner as usual. At 4 p.m. the house surgeon, Mr. Paley, was called to her. She had just vomited, and was said to have "plunged and struggled" a good deal. He found her quite comatose, with marked rigidity of right arm and leg, which were fully extended. No convulsive movements. Pupils equal, not specially dilated, insensible to light. Face pale. Cheeks puffed out during expiration: no stertor. She never regained consciousness, and died quietly at 6.30 p.m.

*Post-mortem examination* on Feb. 11th.—Body well nourished. Right arm rigidly bent at a right angle; left arm rather stiff. Pupils moderately dilated: equal. Urine, drawn off from bladder, pale, somewhat turbid, yielding a distinct trace of albumen.

When the brain was taken out, a small clot was perceived under the arachnoid, just below the tip of the *calamus scriptorius*. On raising the medulla oblongata, the clot was seen to broaden out as it extended into the fourth ventricle. The brain was then turned over. On slicing the left hemisphere, a large clot, resembling black-currant jelly, was found lying in a cavity parallel with, but altogether outside, the lateral ventricle. The clot measured 3 in. in length, 2 in. in breadth, and was at least 1 in. in depth. The walls of the cavity were quite smooth, save at one or two points. It communicated by a small rent with the anterior horn of the left lateral ventricle. The rent was just in front of the corpus striatum, and did not implicate it in the least. Through the opening, a little blood had trickled into the ventricle, where it formed a long, slender, triangular clot. Moreover, a small quantity had made its way through the foramen of Monro, into the right lateral ventricle; also through the third into the fourth ventricle, at whose lower edge it had first been detected.

The original clot, as already stated, lay quite outside the ventricle. It was noticed more particularly that the caudate and lenticular nuclei, together with the internal capsule, were intact; indeed, a thin layer of the external capsule was still attached to the corpus striatum, and formed the inner wall of the cavity occupied by the extravasated blood. The corpus striatum must have been squeezed, but it was not otherwise injured. All attempts to find the exact source of the bleeding failed: no embolon, no aneurism could be detected with the naked eye. The coats of the larger arteries appeared quite healthy.

Lungs œdematous, otherwise normal. The muscular substance of the heart, its lining membrane, and its valves, were healthy, with the following important exception: along the lower edge of the posterior segment of the mitral valve, on its auricular aspect, there was a close-set row of small warty excrescences. At one point, a soft lobulated mass about an inch in length was attached by a relatively narrow pedicle, and hung down into the auriculo-ventricular orifice. Its texture was soft and fragile. Along the corresponding edge of the anterior flap were a few discrete prominences of the tough and warty kind.

The right kidney was much altered. A great part of its substance was made up of yellowish embolic patches, cinctured with narrow zones of hyperæmia. The left kidney contained only one similar patch of small size; the remainder of its tissue was deeply congested. Spleen large, rather soft, containing an infarct as large as a pigeon's egg. Liver and other viscera healthy.

Read backwards, the course of the morbid process in this case is plain enough. Chorea, with its usual attendant, endocarditis; detachment of vegetations from the mitral valve and multiple embolism, causing moderate fever of a remittent type. Two points, however, call for special notice: the mechanism of the hæmorrhage, and the non-discovery of the cardiac lesion during life.

To take the last first. I should never have believed it possible, before this experience, for such very coarse damage to exist in the mitral orifice without making itself manifest by a loud murmur. Had the case not been one of chorea, perfunctory auscultation might reasonably have been suspected. But we have long been in the habit of looking for a mitral murmur in every case of this disease, and of regarding its absence as exceptional. Moreover, the heart was listened to by two persons besides myself, both very competent auscultators. It is just possible that a murmur might have been heard, had the patient been examined in the erect posture; but as she lay in bed, this was not done. Still, the recumbent posture is not usually an obstacle to the recognition of serious mitral disease. Its recognition in the present instance would at once have furnished the real clue to the fever and constitutional disturbance, whose nature, as it was, remained obscure during life.

Next, as regards the hæmorrhage. That a minute aneurism may have existed, though it was not discovered, is, of course, quite possible. And that aneurisms may result from embolism can no longer be questioned, after the evidence brought forward by Dr. Goodhart and others. I believe, however, that

an alternative hypothesis is open to us in the present case. From the position and relations of the clot, it is obvious that the bleeding must have occurred from one of those long branches of the Sylvian artery which make their way upwards outside the lenticular nucleus (*lenticulo-striate* of Charcot). These vessels are all of them *terminal*, i.e. they form no anastomoses with their neighbours between their point of origin and their break-up into capillaries. The impaction of an embolon in a vessel of this kind must needs expose its walls, behind the obstructed point, to a much greater degree of pressure than could occur, under similar circumstances, in an artery ramifying in the usual way. That the lenticulo-striate arteries are ill fitted to resist unusual strain is shown by their liability to rupture in connection with general increase of arterial tension (as in Bright's disease with hypertrophied heart). Accordingly, it may be that in the present instance, the momentary increase of pressure due to the lodgment of an embolon in one of these vessels may have caused a rent behind the obstructed point, large enough to allow of very copious bleeding into a region where, as Broadbent has pointed out, hardly any resistance is offered to extravasation, "the blood only performing a natural dissection in following the line of cleavage between the corpus striatum and external capsule."<sup>1</sup>

<sup>1</sup> "On Ingravescient Apoplexy," *Med. Chir. Trans.* LIX.

## SOME CASES OF NERVE RESECTION AND NERVE STRETCHING.

REPORTED BY HERBERT W. PAGE, M.A., F.R.C.S.

CASE I.—*Neuralgia of the left inferior dental nerve, cured by resection. Pallavicini's operation. Recurrent hæmorrhage. Ligature of common carotid. Various brain symptoms. (Jahresbericht, 1876, der Krankenanstalt Rudolf Stiftung.) Under the care of Dr. Weinlechner.*

A WOMAN, aged 66, had suffered during the past four years from neuralgia in the left inferior dental nerve. At first occasional, the pains gradually increased in frequency, so that latterly she was hardly ever free from paroxysms. These usually occurred spontaneously, but might be brought on by slight causes, such as speaking. They were of a sharp, burning character, and were felt along the whole left body of the lower jaw. In their earlier manifestations the pains spread also to the tongue and to the left upper jaw. All sorts of remedies having failed to give relief, it was decided to resect the nerve. Light pressure on the nerve at its entry into the dental canal was enough to produce pain, though not the terrible suffering which is so common in neuralgia of the third division of the fifth nerve. On July 12th, 1876, the operation within the mouth, following the method of Pallavicini, was performed. The nerve had hardly been isolated and drawn forwards by a thread passed round it before the most profuse bleeding came on. With much difficulty the wound was plugged, and the hæmorrhage stayed. Uncertain what vessel could have been severed, or whether the common carotid should be tied in order permanently to arrest the bleeding, it was determined to proceed with the operation, that being of prime importance. The plug having been carefully removed, there was, to the surprise of the operator, no return of hæmorrhage; and without further trouble 1.5 cm. were resected from the nerve. Neuralgia has never recurred. Immediately

after the operation there was numbness in the left lower jaw, and in the left half of the tongue, the latter being probably due to some unavoidable pressure on the lingual nerve. Half an hour after the operation the bleeding returned, but was easily arrested by plugging. The plug was removed on July 14th, on which day it was noticed that sensation of the tongue was again normal, that of the jaw being still impaired. Everything went on well until July 19th, when, suddenly at 7 A.M., bleeding came on with great fury, and before the House Surgeon could reach her the patient had lost a very large quantity of blood. By careful plugging the hæmorrhage was again stopped, but the woman was much collapsed. The plug was now removed daily, and gradually made smaller to admit of closure of the wound. On July 23rd she was allowed to go out. On July 27th, when she had pretty well regained her strength, the bleeding again came on. The wound again was plugged, this time with the addition of perchloride of iron. The patient was very blanched, and came round but slowly. Once more, on the evening of August 2nd, there was a return of bleeding, and as it seemed probable that the inferior dental itself, near its origin, was the artery wounded, the common carotid was tied on the following morning above the omo-hyoid, with full antiseptic precautions. Bleeding never returned. Immediately after applying the ligature, the left half of the face was paler and cooler than the right. In the evening the patient was weak and wandering. On August 4th, when the wound was first dressed, there was still mental dulness. The right upper extremity appeared numb, and was movable only with effort. No difference was noticed in the legs. On August 5th there was still great hebetude, with advancing "speech-disturbance." There was evident paralysis of the right arm, and to a slight degree of the right side of the face. In the afternoon the right leg was also paralysed. The pulse in the left temporal, although weaker than that of the right, had been distinctly perceptible since the previous evening. The temperature was normal, though the left half of the head was decidedly colder than the right. On August 7th the patient was more sensible. Her ability to move the paralysed limbs was beginning to return, and the right half of the face looked more natural. The aphasia (*sic*), however, was unaltered. On the 8th and 9th she still further improved. The aphasia (*sic*) had now begun to subside, although she could answer only in a whisper, and very slowly. The grasp of the right hand was still much weaker than that of the left. On August 10th it is noted that her speech was tolerably ready, and on the 12th that she was still mentally deficient, having no

remembrance of either the bleeding or the operation. After this date she gradually improved, and on August 26th she was able to walk a little alone, although she slightly dragged her right foot. On September 2nd, when she left the hospital, the patient felt pretty strong, although looking anxious and worn. The arm had fairly regained its strength; the face was almost natural; and there was no difference in the temperature of the two sides of the head. The left half of the tongue was anæsthetic. She seemed to lack mental energy and clearness.

The author remarks that similar results of ligature of the common carotid have been not rarely observed. Schuh described a case in 1848 in which twitchings and convulsions, coming on after ligature of the carotid for repeated bleedings, were followed by weakness of the left limbs, gradually passing into universal hemiplegia. Consciousness was lost. The patient died of pyæmia, and at the *post mortem* the right hemisphere of the brain was found softer and moister than the left. That ligature of the carotid is of itself insufficient to produce such noteworthy symptoms, these and many cases testify. It is probable that precedent and considerable loss of blood, as in his own case and in that of Schuh, has a potent influence in the impairment of nutrition of the hemisphere. The autopsy in the latter case shows, moreover, that a definite organic change in the brain—something more than simple anæmia—must give rise to the predominant symptoms. And this view is supported by the gradual progress, rather than the sudden onset of the paralysis.

A further note of the patient on November 27th records that in many respects she had materially improved, although her memory was still somewhat bad, and her foot was a little dragged in walking.

(Considering that the source of the hæmorrhage was almost certain, the case appears to have been one in which ligature of the external carotid would have sufficed. On this point see an important paper by Mr. Harrison Cripps on the "Treatment of Hæmorrhage from punctured wounds of the Throat and Neck," in vol. lxi. of the 'Transactions of the Royal Medical and Chirurgical Society,' and in which the author, after an exhaustive study of many cases of hæmorrhage, and of the dangers of ligature of the common carotid, urges ligature of the external carotid as a substitute, preliminary at least, for that of the common carotid in cases of emergency, and where simpler means have been tried in vain.)

CASE II.—*Neuralgia of the 3rd Division of the Fifth. Intra-oral resection of the inferior dental, buccal, and lingual nerves, followed by cessation of the neuralgia and the production of sensory and gustatory paralysis on the right fore-half of the tongue.* (*Wiener Medizinische Wochenschrift.* No. 44. 1878.)

This case is abstracted from the 'Jahresberichte der Krankenanstalt Rudolf Stiftung pro 1877,' and was under the care of Dr. Weinlechner in that institution. The wife of a shoemaker, aged 40, eight months before her admission into hospital, on Aug. 7, 1877, began to feel, during a severe cold, a sharp darting pain along the right half of her tongue. Limited at first to the tongue, the pain gradually extended once or twice daily to the hard palate and the right half of the lower jaw. Its frequency increased, and in about six weeks it recurred every five minutes or so, both by night and by day. The pain was aggravated by talking and chewing, and was greatly influenced by changes in temperature. The woman was wasted, and prematurely aged, and she had, in addition to the pain, a copious secretion of saliva. There was no difference in the sense of taste in the two halves of the tongue; nor did pressure on the inferior dental nerve at its entry into its bony canal produce any pain. During the attacks the patient found some relief by constantly rolling her tongue about. In the course of her illness several teeth had been extracted, and other means adopted, without abatement of her suffering. The galvanic and faradic currents when first applied by Dr. Mader very much reduced the pain, which now ceased at night and came on only in talking and eating. The patient herself improved in health, but it was not long before the pain returned in all its original intensity. Its character somewhat changed, for it now became more frequent and more severe in the jaw than in the tongue. The salivary flow returned; there was some flushing of the face, but no painful spot was discoverable. It was therefore resolved to resect portions of the gustatory and inferior dental nerves within the mouth; and this operation, according to the method of Menzel and Pallavicini, was performed on Oct. 10th. The several nerves having been laid bare, a piece 1.5 cm. in length was cut from the inferior dental, and the same length from the buccal nerve, which had been accidentally mistaken for the gustatory. From the lingual itself were removed 2 cm. The attacks of pain at once disappeared, and the following conditions were found on examination: along the inner and outer side of the right half of the lower jaw, on the right half of the tongue and the right half of the floor of the mouth, and of the right side of the chin,

there was complete anæsthesia. The right half of the tongue, moreover, in its anterior parts was insensible to the taste of acid and bitter substances, readily appreciated by the other half. Examination of the sense of taste at the posterior part of the tongue was impossible from swelling of the soft parts. The wound was healed on Oct. 27th, on which day the impairment of taste and of ordinary sensation were the same as directly after the operation. It was only on the left side of the tongue that the patient could feel the rough edge of a glass from which she drank, and she had to take the greatest care in eating to prevent herself from biting her tongue on the right side. When she left the hospital on Nov. 10th, ordinary sensation had somewhat improved, but there was no change in the sense of taste.

As regards this point, a similar observation has been made in the case of a patient whose lower jaw, with a part of the lingual nerve, was removed on account of a large cysto-sarcoma. Professor Brücke, who had several opportunities of examining this patient, considered it incorrect to attribute the impaired taste to the section of the *lingual nerve*, for it is possible that this sense depends rather on the fibres which make their way from the glosso-pharyngeal, by the route of the anastomosis of Jacobson, the small superficial petrosal nerve, and the otic ganglion to the gustatory nerve.

(No mention is made of the chorda tympani nerve, which some observers regard as the gustatory nerve *par excellence* of the anterior area of the tongue. That its fibres were somehow involved is shown by the hypersecretion of the salivary glands—doubtless the submaxillary and sublingual—over whose function the chorda tympani is known to have much influence.)

CASE III.—*Mimic Spasm. Stretching of the Facial Nerve.*  
*Cure.* By Dr. BAUM, Danzig. (*Berliner Klinische Wochenschrift.* Oct. 7, 1878. No. 40.)

A widow, aged 35, of healthy family, though herself delicate, having had much trouble and hard work owing to the illness of her husband and the loss of her only child, had some six years ago a sort of epileptiform attack, thrice repeated on one day. In three weeks she had another—one only, and that the last. Immediately after this she noticed that the left side of her face spasmodically contracted. At first the spasms were mostly limited to the eyelids, but they soon involved the other muscles of the face. In course of time they became more frequent, returning every two or three minutes, and were so violent that, for want of sleep, she was almost worn out.

On admission to the hospital the spasms were incessant and



regular, and lasted with varying intensity for rather more than a minute. With the exception of those of the ear, all the muscles supplied by the left facial seemed to be involved. When the spasms were unusually severe, there were also slight twitchings of the right angle of the mouth. This so much distressed her with a dread of both sides of the face becoming affected, and tonics, galvanism, and other remedies having completely failed, that she willingly submitted to the operation of nerve stretching, which was performed on July 20th of the present year. The facial nerve, after having been, under strictest antiseptic precautions, laid bare near its exit from its foramen, was found to be of a dark red colour, but in no wise thickened. Dr. Baum seized the nerve, and squeezed it pretty firmly with a pair of torsion forceps, while at the same time he drew it out of its bed. Immediately after the operation the left side of the face was completely paralysed. This condition lasted for rather more than half an hour; and there was no return of spasm. The wound healed by first intention, and the patient left the hospital in eight days. When seen shortly before the case was published—or early in October—the scar was quite unnoticeable. There had been no more facial spasm; and, as far as could be judged, the woman was completely cured.

The author fails to discover any precise connection between the facial spasm and the epileptiform attacks, which, from their non-recurrence, he is inclined to regard as of a vertiginous nature, and due to indigestion. He suggests, however, the possibility of some injury to the nerve during the fit which immediately preceded the first twitchings. In a case such as this, where the spasm is mimic and altogether isolated, a central lesion can with certainty be excluded. In this instance, moreover, there was neither constitutional illness nor any peripheral irritation to account for it. The diagnosis was fully confirmed by the appearance of the nerve; and as this itself was at fault, so local treatment was eminently called for. As regards the issue of the operation, he thinks that drawing out of the nerve from the stylo-mastoid foramen, and the substitution thereby of a healthy for an irritated portion of nerve in contact with the bony margin of the canal, must have had undoubted influence in the good result. At the same time it is his conviction that direct squeezing of the nerve-fibres themselves was of much greater value. He cannot help thinking that medical literature might have been enriched with many more successful results of nerve stretching, had operators been rather less tender and delicate in their handling of nerves at the seat of exposure. The fear of paralysis need not be deterrent, for that, should it arise, will quickly pass away.

## CASE OF LOCOMOTOR ATAXY.

BY C. F. NEWCOMBE, M.D.,

*Late Assistant Medical Officer, Lancaster County Asylum, Rainhill.*

BUT few cases of Locomotor Ataxy associated with mental disorder have so far been recorded in this country, and the following case presented features differing in many respects from those I have seen described.

In September 1876, J. K. (single), a Liverpool cotton porter, was brought to the Rainhill Asylum, the medical certificate stating that he was "low-spirited, despondent, obstinate, and taciturn." On examination four days after admission he was found to be in fair bodily condition, of sallow complexion, and with a very melancholy expression. Both his pupils were contracted to a pin's point, and the right was of irregular outline and attached to the cornea. The chest was healthy; his tongue steady and protruded straight; appetite good; urine of dark colour and high specific gravity—no albumen; temperature normal. Patient's gait was jerky, his heel first touching the ground when he put his foot down. With closed eyes he was unable to turn or even to walk without assistance, as he at once began to stagger. Percussion and the application of a hot sponge showed that no single vertebra or skin-area was more tender or sensitive than the rest. On tickling the soles of the feet, reflex action was found to be slow, but much exaggerated when produced. Sensibility in the lower extremities was much diminished.

The patient's account of himself entirely tallied with that contained in the following extract which Dr. Davidson kindly allowed me to make from his Case Book.

J. M. K., æt. 35, single, porter, admitted into the Northern Hospital, Liverpool, Sept. 18, 1874 (under the care of Dr. Davidson). Patient's complaint is pretty evident, consisting of ataxic symptoms which have the following history.

About the middle of January of the present year he first felt his feet cold and a sense of numbness in them; shortly after, he began to experience difficulty in locomotion which made

him give over work. In about a month he began to have a springing sensation in the soles of his feet, so that he tended to fall forward, and felt as if he was walking on poultices. About the end of January, when going to church he noticed that he had great difficulty in going up the steps. At present he does not find it much more difficult to come up than go down stairs, if the steps are broad, but when they are narrow he finds the truth of Virgil's line

"Sed revocare gradum, superasque evadere ad auras  
Hoc opus, hic labor."

He was several months under medical treatment, and took various patent medicines, such as "Blair's pills," and £5 worth of Dr. Swedor's "Vital Regenerator." Has had 5 weeks' application of the faradic current applied six times a week for about  $\frac{1}{2}$  hour each day. Has also had many salt-water baths.

*Previous History.*—About 15 years ago he had a chancre and bubo, some months later a gonorrhœa, and shortly afterwards had rheumatic fever. Has always otherwise been healthy. Never had skin eruption, nor alopecia, nor sore throat.

Thinks he has been falling off in flesh of late; at present his muscles are soft and flabby. Height about 5 ft. 6 in., weight never exceeded 10 stone.

*Locomotor System.*—He walks with feet widely apart, carries his limbs hurriedly forward and away from the mesial line. In throwing his limbs forward his feet are powerfully extended, the heel being brought rapidly down, and the rest of the foot following with a flapping motion. He can walk pretty well on a base of 10 inches. His locomotor difficulties are increased by closing his eyes, and then when standing he has a swaying movement. He has great muscular power, and can resist with much force any interference with his limbs. His legs when at rest are often affected with irregular involuntary movements, and even the strongest efforts of patient's will are unable to overcome these spasmodic muscular contractions. He can distinguish between heat and cold, but the transmission of thermal impressions is delayed, those of heat being most so; cold is felt from  $1\frac{1}{2}$  to 2 seconds after impression is made, and heat felt about 3 seconds after limb is touched. The tactile sensibility is much diminished in the feet, and even painful impressions are not acutely felt. There is no special tender spot along the spine, and hot and cold sponges are felt equally throughout.

The muscular power of his arms, as tested by the dynamometer, is good, and in them the power of co-ordination is perfect. Has occasional diplopia, but this followed an injury to and operation on the right eye 20 years ago. His body is marked with small cicatrices, the result of small-pox, which he

had when 5 years old. Both pupils are greatly contracted and equally so.

*Nov. 4th.*—Examination of the heart reveals slight roughening with the first sound at the base.

There is some sickness, anorexia, and loose bowels ; pulse 100.

*Nov. 9th.*—The cardiac phenomena referred to in last note are perhaps more distinct to-day. The apex is in fifth interspace, and in line of nipple. Transverse measurement 4 inches.

The cardiac dulness extends up the sternum as high as the junction of the manubrium with the body of the sternum. Over this narrow strip of dulness and at the base there is a roughening of the first sound, of a probably pericardial origin. The second sound is also somewhat harsh and prolonged.

Appetite bad, bowels rather loose, pulse weak.

*Nov. 28th.*—Patient feels anxious to leave to-day, and consequently is discharged.

After leaving the hospital he again tried the effects of galvanism and remedies prescribed by herbalists and others, to whom he despairingly applied for relief, but at last, finding his case considered a hopeless one, he seems to have fallen into a despondent state of mind, his temper became irritable and suspicious, and finally his friends had to remove him to the workhouse on account of his obstinacy and general unmanageableness.

He related his story slowly, in plaintive, melancholy tones, but without hesitation or tremor of the voice ; appeared to have a good memory for the dates and details of both recent and remote occurrences, and when roused answered all questions readily and accurately, although when left to himself he was always absorbed in the contemplation of his own ailments.

In October it was noted that he was still very depressed and fond of solitude, that he had frequent outbursts of crying and sobbing, and at times expressed great alarm lest castration should be practised as a remedy for his disease. Nocturnal emissions occurred frequently. Patient wrote several times to his friends, his writing being of average firmness, his letters well formed and no words omitted. He had great power in his arms, and made himself useful by blacking all the boots in his ward. About a month later he complained of the pains and aches in his lower limbs being much worse, and attributed them to the action of workmen in the ward who "were killing him with their raps and taps." At night he occasionally became violent, the snoring of his neighbours sounding in his ears as the voices of "Roman Catholics telling him to curse his Queen and the Protestant religion."

For many months after this his mental condition remained stationary, although his bodily symptoms were steadily pro-

gressing. He complained of double vision, though the axes of his eyes were to all appearance parallel. He began to have suspicions as to his food being drugged, and frequently struck other patients, under the idea that the shocks and starts he felt in his extremities were caused by them. In September 1877, on three several occasions while he was sitting quietly on his chair, his legs became strongly convulsed without any previous warning, causing him to fall on the floor, where he lay for two or three minutes on his back, ineffectually endeavouring to check the extravagant movements of his limbs. His articulation had now become slower and rather tremulous, but mastication and deglutition were well performed. His handwriting remained firm and distinct, but words and letters were omitted. Double vision had ceased, but his sight grew much weaker. His pupils continued, as from the first, minutely contracted. He complained of having to micturate very frequently, but retained full control over his sphincters. Attacks of emotional excitement grew more numerous and prolonged, and often occurred when he was alone. He also held long conversations with imaginary personages, who used, he said, very bad language. At the beginning of the present year he began to lose flesh and to grow weaker; his memory was much more impaired; he appeared to be entirely engrossed in himself; his tones were more querulous, and he used to complain that twenty ounces of chloroform were given him by the night attendant every night. The fulminating pains were as strong as ever, but sensibility in his lower extremities had decreased, and, although he was quite unable to walk without steadying himself by walls and furniture, and even when his feet flew out laterally to the danger of passers by, his hands and arms were still only affected to a very slight degree. In March he was found to be very much weaker; he had retention with incontinence of high-coloured ammoniacal urine, which had to be regularly drawn off, when he complained that French semen was being substituted for his own. Introduction of the catheter was always painful. Speech was more affected, swallowing at times difficult.

From this time until his death on April 16th, 1878, he rapidly lost flesh, but was always able to feed and raise himself. Flexion of his lower limbs could be resisted with some power until the day before his death. Whenever the weight of the bed-clothes was taken off his legs strong spasmodic jerking commenced, and was continued until the clothes were replaced. The temperature in the axilla, which had averaged about 100° for some days, did not rise to any great extent before his death, and his lungs remained fairly healthy.

At the autopsy, made twenty-seven hours after death, the

body was found to be much emaciated. The calvarium was dense, and the dura mater adherent to it. The arachnoid was slightly thickened, the vessels of the pia mater nearly empty. The membranes were adherent only over the tips of the temporo-sphenoidal lobes. There was some wasting of the convolutions bordering on the fissure of Rolando, and a large quantity of fluid escaped on removal of the membranes and on opening the ventricles. The lining membrane of the lateral ventricles was granular, the arteries at the base of the brain normal. The brain substance was firm and healthy-looking, and the weight of the whole organ fifty-four ounces. On examination of the recent spinal cord, the pia mater was found to be congested; the cord itself wasted, and of a dirty greyish hue on removal of the membranes. On section extensive grey degeneration was distinctly seen in the posterior regions, especially in the lower dorsal and the lumbar portions of the cord.

On microscopical examination of sections made by Dr. Ashby, of the Liverpool School of Medicine, after hardening in bichromate of ammonia, the usual changes of the structure of the cord were seen, but in a very advanced stage. Corpora amy-lacea were numerous; and the large multipolar cells of the posterior cornua of grey matter had almost disappeared in the lower part of the cord, while those in the anterior were much diminished in size and number.

*Remarks.*—It was pointed out by Dr. Nichol, in the first volume of the 'West Riding Asylum Medical Reports,' that the mental symptoms accompanying locomotor ataxy are, in the large majority of instances, marked by delusions of an exalted character, and in six out of the eight cases he describes, or quotes, these were present. In the case of J. McK., however, there seems to have been no period during which he experienced pleasurable emotions dependent upon the feeling of power. On the contrary, great depression due to the sense of growing weakness characterised his history throughout.

Most of his hallucinations and delusions, too, were apparently due to perverted sensations; the brain receiving abnormal impressions from and through the diseased sensory-tracts, and referring them wrongly to peripheral influences.

In addition to the slight nature of the changes that were discoverable in the brain itself, it is worthy of remark that this organ weighed nine ounces more than the average weight of the brain in 197 cases of general paralysis, and ten ounces more than the average weight of the brain in 700 cases of all varieties of insanity, as recorded by Mr. Crochley Clapham, in vol. vi. 'West Riding Asylum Medical Reports.' 'The Weight of the Brain in the Insane,' by C. Clapham, L.R.C.P., &c.

## Abstracts of British and Foreign Journals.

### Progressive Amyotrophic Bulbar-Paralysis, and its Relations to Symmetrical Sclerosis of the Lateral Columns.

E. LEYDEN. (*Arch. f. Psych.*, viii. p. 641.)—In this paper the author gives a short account of the history of progressive bulbar-paralysis, and discusses its symptomatology, pathological anatomy, and pathological physiology. Its pathological anatomy was almost unknown to Duchenne, who thought it a peripheral affection of the muscles. Bärwinkel argued that as the faradic irritability of the muscles remained intact, the lesion was probably central and seated in the medulla. Then Wachsmuth, from an analysis of the symptoms, concluded that the lesion was in the medulla in the vicinity of the nuclei of Stilling, and was of a similar nature to the grey degeneration of the posterior columns seen in tabes. Trousseau next observed macroscopically the increased consistence of the medulla, and he and Wachsmuth found fatty degeneration in the roots of the hypoglossal, vagus, and facial nerves. Charcot, Duchenne and Joffroy, and the author almost contemporaneously recognised in addition to the lesions of the muscles and nerve roots, degeneration of the motor strands of the spinal cord, atrophy of the grey matter of the anterior cornua, and disappearance of the multipolar ganglion-cells in these cornua and in Stilling's nuclei.

The following is a summary of the pathological appearances observed in the five cases that were under the author's care, and two of which are very carefully reported in this paper:—(1) The muscular atrophy was most marked in the tongue, then in the lips, and next, in the hand and fore-arm. It was a simple, unequally-distributed atrophy, some fibres being very small, others almost of normal breadth. (2) The motor nerves were degenerated in every part of their course, from origin to termination. The nerve fibres themselves were affected, the interstitial tissue and the nerve-sheath remaining normal. Two stages of the process

were noticed,—first, fatty degeneration; second, sclerotic atrophy. The nerves presented similar appearances to those observed in the motor nerves after Wallerian atrophy. (3) The posterior columns, posterior nerve-roots, and posterior cornua of the cord were unaltered (in one case there was a thin strip of sclerosis on the inner side of each posterior column). There was marked degeneration of both pyramidal strands; and the remaining parts of the anterolateral columns, comprising the intraspinal motor fibres of Flechsig, were degenerated, but in a much less degree. The degenerative process could be detected in the decussation of the pyramids, the anterior pyramids, and the pyramidal fibres of the lower half of the pons. It could not be discovered in the upper part of the pons. Microscopic examination of the affected strands showed a considerable diminution in the number of nerve fibres, and those that remained were atrophied. There was very pronounced atrophy of the large ganglion-cells of the anterior cornua, especially in the region of the cervical enlargement. Charcot, Duchenne, and others regard these ganglion-cells as trophic centres for the motor fibres, and attribute the muscular atrophy to disorganisation of them. The cells of the lateral portions of the grey matter and of Clarke's nuclei were always intact. There were fewer nerve fibres in the grey matter than there are normally. In the medulla oblongata the nerve-nuclei of Stilling were atrophied, and presented appearances analogous to those found in the anterior cornua. These changes were beautifully seen in the nuclei of the hypoglossal. In the nuclei of the facial, vagus, and accessorius they were not nearly so marked.

The distribution of the principal lesion is remarkable: it is symmetrical, and, though wide-spread, is limited to the *motor* cells and fibres of the spinal cord and medulla oblongata. It is an instance of a lesion attacking a particular physiological system of fibres (Flechsig). The morbid process is essentially a chronic parenchymatous degeneration, commencing in the nerve cells and spreading along the fibres connected with them, any changes that there are in the neuroglia being secondary. In far advanced cases, the lesion had the character of sclerosis; in less advanced cases, it was very similar to the descending degeneration of Türck. The process does not always commence in one place.

After noticing the correspondence between the symptoms and the lesion in the cases described by him, the author draws attention to the points on which he differs with Charcot. According to the latter, the progressive muscular atrophy is preceded by paralysis;



according to the former, the atrophy is primary, and is never preceded by paralysis. Again, the two authors differ as to the type of the muscular affection. Charcot describes a tonic (spastic) paralysis with rigidity, spasm, and contracture; Leyden, a paralysis with relaxation (atonic paralysis). Charcot's view,—that contracture and muscular spasms are caused by sclerosis of the lateral columns,—is adversely criticised, and the conclusion arrived at, that degeneration of the lateral columns is not necessarily followed by contracture and rigidity, and that therefore these symptoms are not pathognomonic of the lesions in question. Indeed, the same symptoms have been found by Leyden, Kilian, and by Charcot himself in cases of diffuse sclerosis (diffuse chronic myelitis) of the whole spinal cord.

In an appendix to the paper, the author gives abstracts of the cases of progressive amyotrophic bulbar-paralysis (including his own five cases, twelve in number) that have been reported. Then follow three cases in which the course of the disease was somewhat abnormal, and, lastly, two in which there was no lesion of the lateral columns.

**A New Theory of Locomotor Ataxia.**—In a preliminary communication to the *Centralblatt für Med. Wissensch.*, Dec. 14, 1878, Dr. Takács gives the following conclusions as the results of clinical and anatomical investigation:—

(1.) The grey degeneration of the posterior columns which is almost always found in tabes is a secondary process; the primary affection is an atrophy of the posterior nerve roots and posterior cornua, or a *meningitis posterior*.

(2.) The most constant of the derangements of sensibility is the very slow conduction of sensory impressions. This is never absent, whereas anæsthesia (hypæsthesia) is often absent.

(3.) The posterior columns conduct tactile impressions only; and the grey substance normally conducts only sensations of pain (Schiff). But after pathological alterations of the posterior columns the grey substance can act vicariously for them, and conduct tactile impressions (Friedrich). The rapidity of conduction is now, however, diminished.

(4.) In a normal movement, the muscles contract, not with a jerk, but gradually and with an intensity varying continually until the aim of the movement is accomplished. This every-varying intensity of contraction is produced by the ever-varying intensity of the impulses that are sent from the co-ordinating centres to the muscles along the motor nerves, the intensity of the impulses,

again, being regulated by the impressions the sensory nerves convey of the phase of muscular movement attained.

(5.) If, however, the conduction of these controlling sensory impressions is delayed, the impulses excited by them in the co-ordinating centres will likewise be delayed, and, as a consequence, the muscle from an already attained phase of contraction will sink back to an earlier and lower phase, &c. . . . in other words, the movement will be ataxic.

(6.) In tabes, the delay in sensory conduction is due to the fact that the function of the degenerated posterior columns, which normally are rapid conductors, is performed by the more slowly conducting grey substance.

(7.) Where the posterior columns alone are degenerated, there is only ataxia; but where the posterior cornua and posterior roots are also affected, in addition to the ataxia there is anæsthesia (hypæsthesia).

[This scarcely seems to agree with conclusions 1 and 2.]

**Progressive Muscular Atrophy.** LICHTHEIM. (*Arch. f. Psych.* viii. p. 521, abstract by BERNHARDT in *Centralblatt*, 1878, No. 50.)—In a case of progressive muscular atrophy observed by Lichtheim the muscles presented the typical histological appearances of muscular atrophy, but the nerves, peripheral and central, were perfectly normal. A most careful microscopic examination failed to detect either atrophy of the large ganglion-cells of the anterior cornua, or alteration in the cervical ganglia of the sympathetic, or in the anterior roots of the spinal nerves. Lesion of the ganglion-cells of the anterior cornua, therefore, is not a necessary condition in cases of progressive muscular atrophy; and, as Friedreich remarks, in many, perhaps in most cases, the spinal changes are to be looked upon as merely secondary, the primary affection being the changes in the muscles. The nearly allied pseudo-hypertrophic paralysis, Lichtheim agrees with Charcot, Cohnheim, and Eulenburg in regarding as a peripheral affection. As primary affections of the anterior cornua, he includes only (1) acute atrophic spinal paralysis (infantile paralysis, and the analogous affection in adults), (2) subacute atrophic spinal paralysis (subacute anterior spinal paralysis of Duchenne), and (3) chronic atrophic spinal paralysis.

**Multiple Cerebro-Spinal Sclerosis.** DR. CLAUS. (*Allgem. Zeitschr. f. Psych.*, 1878, p. 335.) Claus reports a case in which

after death multiple cerebro-spinal sclerosis was found. General paralysis had been diagnosed, the diagnosis being founded on the mental condition of the patient, his defective articulation, which resembled that observed in general paralytics, and the course of the paralysis, which was at first remittent, but afterwards became progressive and general. Further, that the spinal cord was specially affected seemed evidenced by the existence of sensations of tingling, &c., in legs and arms, the diminished power of localising tactile sensations, and the increased reflex excitability. But the idea of cerebro-spinal sclerosis was not entertained on account of the absence of some of the more typical symptoms observed in that disease, *e.g.* the peculiar scanning articulation, diplopia, nystagmus, rigidity and contracture of muscles. Tremors were only observed once, and then but for a short time in the right leg. There was slight weakness of sight, and hearing was somewhat impaired on the left side (in 41 cases of cerebro-spinal sclerosis, the records of which were examined by Claus, hearing was only implicated twice). This case shows that what are described as the characteristic symptoms of cerebro-spinal sclerosis may be absent or very little marked during life, and yet after death very distinct and wide-spread evidence of its presence be found. At the *post-mortem*, in addition to the nodules of sclerosis, there was adhesion of the pia mater over the frontal and the ascending frontal convolutions.

**The Influence of the Central Nervous System on the Irritability of the Motor Nerves.** TH. RUMPF. (*Arch. f. Psych.* viii., p. 567, and *Centralblatt*, 1878, No. 50.)—The author experimented on frogs with the ascending current. He found (1) that the muscular contraction produced by breaking of the current comes on later if the motor nerve is connected with the spinal cord than if it is separated from it. The connection between the nerve and the cord was severed by simple section of the nerve, by destruction of the part of the cord from which the nerve arose, and by the destructive action of ammonia. The last method has the advantage of destroying the conducting power of the nerve without causing any muscular contraction from its irritation. Decapitation did not affect the results of these experiments. (2) After the application of cold to the spinal column for fifteen to thirty minutes the breaking contraction is still later in its appearance. (3) The same effect follows separation of the head and vertebral column from all other parts of the body, with the excep-

tion of the leg operated on, during the first fifteen to twenty minutes. (4) These results are not due to reflex action through the sensory roots, for they were obtained after the use of chloral, and after section of the sensory roots. The author concludes that in motor nerves connected with the spinal cord, influences are at work which are not observed in motor nerves separated from the cord, one indication of the existence of these influences being the modification of the electrical irritability of the nerve. Whether these influences are identical with those that regulate the tonus and nutrition of nerves and muscles, it is impossible to say. Erb thinks that the motor strands in the central nervous system are distinct from the trophic apparatus, since there are cases of central paralysis, with and without atrophy; and, on the other hand, cases of atrophy and degeneration of muscles with central lesion, and yet no paralysis and no loss of the irritability of the nerves supplying the muscles: and, further, since a muscle can degenerate while its nerve remains intact, he thinks that both nerve and muscle have a separate trophic apparatus.

Clinical observations tend to show that in man also the muscular contraction produced by breaking an ascending current comes on at an earlier period if the nerve is not connected with the spinal cord. Two cases of peripheral paralysis of the radial nerve are recorded, in both of which there was a heightened irritability of the injured nerve at the break of an ascending current. As the paralysis disappeared, the nerve returned to its normal state. In cases where the paralysis has existed for a considerable time, and where the electrical irritability of the nerve is much diminished, the heightened irritability just mentioned is no longer found.

In the secondary muscular atrophy that so often follows joint affections, the electrical irritability of the nerve is less for currents of all kinds. Rumpf agrees with Paget and Valtat in regarding this and the muscular atrophy as a reflex phenomenon due to an arrestment of the action of the spinal cord by the strong sensory impulses that reach it from the affected part.

W. J. DODDS, M.B., D.Sc.

**Pathological Lesions versus Motor Centres.**—Dr. Dario Maragliano has reprinted his paper from the *Rivista di Freniatria*, one moiety of which we have noticed in a previous number (Part III.). He pursues his argument in favour of the localisation of motor centres in the hemispheres, and gives at the end of his pamphlet the following deductions:—

(a.) The movements resulting from excitation of the grey matter of the cortex cerebri cannot be owing to diffused currents acting on the underlying parts.

(b.) The hypothesis of Schiff, who regards the excitable points of the cortex as centres of tactile sensation, and that of Hitzig and Nothnagel, who make the derangements of motion following disturbances of the vascular centres to be dependent upon an alteration of the muscular sense, are inadmissible, especially when we consider the results of clinical observation.

(c.) The hypothesis of Brown-Séquard, who denies the possibility of localising in the cortex or any other part of the brain functions of any kind, besides being a denial of all scientific progress, is contradicted by experimental and clinical results, and the facts brought to sustain his thesis are not of a kind to destroy the doctrine of localisation.

(d.) Those points of the cerebral cortex in which irritation is followed by motor phenomena, and whose destruction is followed by paralysis, ought to be regarded as true voluntary motor centres, capable, without the necessary intermediary of any other centres, of arousing the functions of the various muscular apparatus. These peculiar properties of the centres of the cortex and their independence of the ganglia of the base of the brain are attested both by the results of clinical and experimental observation and by anatomical researches, by the descent of the fibres of the brain and their degeneration following upon lesions of the motor zone of the cortex, and the occurrence of atrophy of portions of the cortex which have followed amputation or arrest of the development of a limb, as well as by the results of microscopic search, demonstrating the peculiarity of the structure of the motor area.

(e.) In man, the compensation for a lesion of the cortex, even of some extent, is possible from the opposite hemisphere, especially when the left one is uninjured; and this is effected, mainly if not always, through means of the fibres which go direct from the healthy hemisphere to the same side.

**Diagnosis of Lesions of the Cortex Cerebri.**—Dr. Maragliano (*Sulla Sintomatologia delle Lesioni Corticali della Zona Motrice, Reggio Emilia*, 1878) argues that we have means of distinguishing a lesion of the cortex cerebri from one of some other parts of the brain. He thinks that convulsions arising from injuries to the cortex are generally unilateral or restricted to one limited group of muscles. After the cortex cerebri, the centrum ovale is thought to

be the only one that gives origin to localised fits; but we are in want of instances of such fits resulting where the overlying grey matter is unaffected. After diligently searching medical literature, he cannot find a single example of partial epilepsy which is not owing to a lesion of the cortex. The loss of consciousness comes on late or not at all, and the paralysis appears gradually and is circumscribed, or there is hemiplegia of one side coming on gradually. The paralysis is soon followed by contractions, or is associated with aphasia, having the character of amnesia or verbal ataxia, or of verbal paralysis. There is a slight rise of temperature in the paralysed limb, and if there be a localised pain in the one part of the head, either spontaneous or brought out by percussion, then the diagnosis of a cortical lesion cannot be doubtful.

**Ergot in Insanity.**—Dr. Enrico Toselli (*Archivio Italiano*, Settembre 1878) has a long paper on the effects of ergot of rye in the treatment of mental derangement. He thinks that this drug produces cerebral anæmia, its action being the reverse of nitrite of amyle. In fact, he has found by experiment that, contrary to the opinion of Schüller, the cerebral vessels contracted by ergot may be dilated by the inhalation of nitrite of amyle. Brown-Séquard demonstrated that the primary effect of ergot was the contraction of the blood-vessels in all the organs in the body, as well as the contraction of the fibres of the uterus. Vokes obtains favourable results in treating hemicrania; Silva, in the treatment of cerebral hyperæmia; Crichton Browne, in the congestive form of mental alienation in recurrent mania, in chronic mania with lucid intervals, and in epileptic mania. Dr. Toselli found it of great use in treating serous diarrhœa, a frequent complication of dementia, especially in the paralytic form. In administering it for this purpose he observed that his patients passed out of the state of sleeplessness, and that their mental faculties were less obtuse. He either used the aqueous extract of the *Secale cornutum*, or the *ergotin Bonjean*, given twice during the night in doses of from 50 centigrammes up to as much as 4 grammes. He found that ergotin acted most quickly and surely in the form of hypodermic injection. Ergot diminishes the frequency of the pulse, contracts the vessels, augments the pressure of the blood, and lowers the temperature. Digitalis has more power in moderating the action of the heart, whereas ergotin has a greater effect upon the blood-vessels and in diminishing the temperature. Sometimes ergotin acts as a diaphoretic and diuretic. Sometimes the therapeutic effects have not appeared with a large dose, and

only manifested themselves when it was reduced. Sometimes the calmative effect following the use of ergotin lasted as long as a month. Toselli used the drug in thirty cases and found the most benefit from it in paralytic insanity, in chronic mania, and in dementia accompanied by agitation, insomnia, hallucination of the senses, especially when these symptoms accompany melancholia and hypochondria. He does not pretend to have cured any case of insanity with ergotin, though he thinks it may arrest the course of general paralysis.

W. W. IRELAND.

### The Spinal Ganglia and Spinal Cord of the *Petromyzon*.

By S. FREUD. (*Sitzber. d. K. Acad. d. Wissensch. Wien. Bd. 78*).—The spinal ganglia of *Petromyzon* and *Amocetes* are, on account of their simplicity and the small number of their elements, well adapted for study in comparison with those of higher animals, and many questions of importance may thus receive a definite solution. A part of the posterior roots—about two-thirds—remains unconnected with ganglion cells. The ganglion cells are bipolar; an appearance of tripolar cells is caused by the splitting of one of the processes in some cases. Divisions of the fibres are not common. Spinal ganglia and posterior cornu form a connected whole in reference to the posterior roots. Such fibres as are clearly connected with cells in the spinal ganglia, in all probability do not enter cells in the posterior cornua, and *vice versa*.

The spinal pia mater—in gold preparations—exhibits a delicate network of varicose nerve-fibres, which do not seem to have any relation to the blood-vessels.

### Proliferations of the Ependyma Ventriculorum. T. WEISS.

(*Wiener Med. Jahrbücher*, 1878.)—The Ependyma is composed first of a layer of epithelial cells which everywhere in all the ventricles, and also in adult life, are furnished with cilia. Underneath the epithelial layer there is a layer of closely interwoven connective tissue, with comparatively few, but still very distinct cells with numerous processes. The deep boundary of the ependyma is marked by vessels which run parallel with the ventricular surface. Proliferations of the ependyma and hydrocephalic effusions into the ventricles do not always go together. The excrescences are developed out of the primitive connective tissue corpuscles. First roundish cells without processes appear, which afterwards throw out processes, and thus become the knots of a

connective tissue net-work. These connective tissue corpuscles are always found in greatest abundance round the deep-lying vessels. [The author overlooks the very likely hypothesis that they may have migrated from the vessels.] The excrescences are not always covered with epithelium. On the contrary, epithelial cells are very often found in their interior. This may be accounted for in two ways; either that the proliferating connective tissue breaks through the epithelial covering, and then advances farther on the ventricular surface, or that two neighbouring nodules fuse with each other.

H. OBERSTEINER.

**Hystero-Neuroses.**—DR. ALLAN McLANE HAMILTON (*Saint Louis Medical and Surgical Journal*, January, 1879) believes that many cases of so-called organic neuroses are examples of unusual hysteria, and he considers that the development of real organic disease after a continued hysterical condition is quite possible. In one particular instance he was assured that a degeneration of the lateral column had followed hysterical paralysis and contracture of a lower extremity. In the diagnosis of hysterical from organic conditions he calls attention to the absence in the former of symptoms which always accompany the latter. One feature, the absence of which is marked in hysterical simulations, is cranial nerve paralysis, with its numerous superficial manifestations. Dr. Hamilton brings forward one case in which the hysterical condition, accompanied by epilepsy, passing into the status, headache, erythema and cerebral vomiting, closely simulated syphilitic brain disease. The symptoms suddenly disappeared, but some of them returned along with an hysterical attack at the patient's next period. In this case there was false perception of colour—a symptom which Dr. Hamilton considers suggestive of hysteria. The writer also comments on the repeated observation of hysterical symptoms in cases of organic brain disease.

R. LAWSON, M.D.



# BRAIN.

JULY, 1879.

Original Articles.

## PSYCHOMETRIC EXPERIMENTS.

BY FRANCIS GALTON, F.R.S.

PSYCHOMETRY, it is hardly necessary to say, means the art of imposing measurement and number upon operations of the mind, as in the practice of determining the reaction-time of different persons. I propose in this memoir to give a new instance of psychometry, and a few of its results. They may not be of any very great novelty or importance, but they are at least definite, and admit of verification; therefore I trust it requires no apology for offering them to the readers of this Journal, who will be prepared to agree in the view, that until the phenomena of any branch of knowledge have been subjected to measurement and number, it cannot assume the status and dignity of a science.

The processes of thought fall into two main categories: in the first of these, ideas present themselves by association either with some object newly perceived by the senses or with previous ideas; in the second process, such of the associated ideas are fixed and vivified by the attention, as happen to be germane to the topic on which the mind is set. In this memoir I do not deal with the second process at all, so I need not speak more in detail concerning it, but I address myself wholly to the first. It is an automatic one; the ideas arise of

their own accord, and we cannot, except in indirect and imperfect ways, compel them to come.

My object is to show how the whole of these associated ideas, though they are for the most part exceedingly fleeting and obscure, and barely cross the threshold of our consciousness, may be seized, dragged into daylight, and recorded. I shall then treat the records of some experiments statistically, and will make out what I can from them.

I should be glad if the reader would refer to an article written by me in the 'Nineteenth Century' of last March, which was based on the observations I am about to describe. It travels somewhat further afield than the present memoir, but does not enter so much into details.

When we attempt to trace the first steps in each operation of our minds, we are usually baulked by the difficulty of keeping watch, without embarrassing the freedom of its action. The difficulty is much more than the common and well-known one of attending to two things at once. It is especially due to the fact that the elementary operations of the mind are exceedingly faint and evanescent, and that it requires the utmost painstaking to watch them properly. It would seem impossible to give the required attention to the processes of thought and yet to think as freely as if the mind had been in no way preoccupied. The peculiarity of the experiments I am about to describe is that I have succeeded in evading this difficulty. My method consists in allowing the mind to play freely for a very brief period, until a couple or so of ideas have passed through it, and then, while the traces or echoes of those ideas are still lingering in the brain, to turn the attention upon them with a sudden and complete awakening; to arrest, to scrutinise them, and to record their exact appearance. Afterwards I collate the records at leisure, and discuss them and draw conclusions. It must be understood that the second of the two ideas was never derived from the first, but always directly from the original object. This was ensured by absolutely withstanding all temptation to reverie. I do not mean that the first idea was of necessity a simple elementary thought: sometimes it was a glance down a familiar line of associations, sometimes it was a well-remem-

bered mental attitude or mode of feeling, but I mean that it was never so far indulged in as to displace the object that had suggested it, from being the primary topic of attention.

I must add, that I found the experiments to be extremely trying and irksome, and that it required much resolution to go through with them, using the scrupulous care they demanded. Nevertheless, the results well repaid the trouble. They gave me an interesting and unexpected view of the number of the operations of the mind, and of the obscure depths in which they took place, of which I had been little conscious before. The general impression they have left upon me is like that which many of us have experienced when the basement of our house happens to be under thorough sanitary repairs, and we realise for the first time the complex system of drains and gas- and water-pipes, flues, bell-wires, and so forth, upon which our comfort depends, but which are usually hidden out of sight, and of whose existence, so long as they acted well, we had never troubled ourselves.

The first experiments I made were imperfect, but sufficient to inspire me with keen interest in the matter, and suggested the form of procedure that I have already partly described. My first experiments were these. On several occasions, but notably on one when I felt myself unusually capable of the kind of effort required, I walked leisurely along Pall Mall, a distance of 450 yards, during which time I scrutinised with attention every successive object that caught my eyes, and I allowed my attention to rest on it until one or two thoughts had arisen through direct association with that object; then I took very brief mental note of them, and passed on to the next object. I never allowed my mind to ramble. The number of objects viewed was, I think, about 300, for I have subsequently repeated the same walk under similar conditions and endeavouring to estimate their number, with that result. It was impossible for me to recal in other than the vaguest way the numerous ideas that had passed through my mind; but of this, at least, I was sure, that samples of my whole life had passed before me, that many bygone incidents, which I never suspected to have formed part of my stock of thoughts, had been glanced at as objects too familiar to awaken the attention.

I saw at once that the brain was vastly more active than I had previously believed it to be, and I was perfectly amazed at the unexpected width of the field of its everyday operations. After an interval of some days, during which I kept my mind from dwelling on my first experiences, in order that it might retain as much freshness as possible for a second experiment, I repeated the walk, and was struck just as much as before by the variety of the ideas that presented themselves, and the number of events to which they referred, about which I had never consciously occupied myself of late years. But my admiration at the activity of the mind was seriously diminished by another observation which I then made, namely that there had been a very great deal of repetition of thought. The actors in my mental stage were indeed very numerous, but by no means so numerous as I had imagined. They now seemed to be something like the actors in theatres where large processions are represented, who march off one side of the stage, and, going round by the back, come on again at the other. I accordingly cast about for means of laying hold of these fleeting thoughts, and, submitting them to statistical analysis, to find out more about their tendency to repetition and other matters, and the method I finally adopted was the one already mentioned. I selected a list of suitable words and wrote them on different small sheets of paper. Taking care to dismiss them from my thoughts when not engaged upon them, and allowing some days to elapse before I began to use them, I laid one of these sheets with all due precautions under a book, but not wholly covered by it, so that when I leant forward I could see one of the words, being previously quite ignorant of what the word would be. Also I held a small chronograph, which I started by pressing a spring the moment the word caught my eye, and which stopped of itself the instant I released the spring; and this I did so soon as about a couple of ideas in direct association with the word had arisen in my mind. I found that I could not manage to recollect more than two ideas with the needed precision, at least not in a general way; but sometimes several ideas occurred so nearly together that I was able to record three or even four of them, while sometimes I only managed one. The second ideas were,

as I have already said, never derived from the first, but always direct from the word itself, for I kept my attention firmly fixed on the word, and the associated ideas were seen only by a half glance. When the two ideas had occurred, I stopped the chronograph and wrote them down, and the time they occupied. I soon got into the way of doing all this in a very methodical and automatic manner, keeping the mind perfectly calm and neutral, but intent and, as it were, at full cock and on hair trigger, before displaying the word. There was no disturbance occasioned by thinking of the imminent revulsion of the mind when the chronograph was stopped. My feeling before stopping it was simply that I had delayed long enough, and this in no way interfered with the free action of the mind. I found no trouble in ensuring the complete fairness of the experiment, by using a number of little precautions, hardly necessary to describe, that practice quickly suggested, but it was a most repugnant and laborious work, and it was only by strong self-control that I went through my schedule according to programme. The list of words that I finally secured was 75 in number, though I began with more. I went through them on four separate occasions, under very different circumstances, in England and abroad, and at intervals of about a month. In no case were the associations governed to any degree worth recording, by remembering what had occurred to me on previous occasions, for I found that the process itself had great influence in discharging the memory of what it had just been engaged in, and I of course took care between the experiments never to let my thoughts revert to the words. The results seem to me to be as trustworthy as any other statistical series that has been collected with equal care.

On throwing these results into a common statistical hotch-pot, I first examined into the rate at which these associated ideas were formed. It took a total time of 660 seconds to form the 505 ideas; that is at about the rate of 50 in a minute or 3000 in an hour. This would be miserably slow work in reverie, or wherever the thought follows the lead of each association that successively presents itself. In the present case, much time was lost in mentally taking the word in, owing to the quiet unobtrusive way in which I found it necessary

to bring it into view, so as not to distract the thoughts. Moreover, a substantive standing by itself is usually the equivalent of too abstract an idea for us to conceive it properly without delay. Thus it is very difficult to get a quick conception of the word "carriage," because there are so many different kinds—two-wheeled, four-wheeled, open and closed, and all of them in so many different possible positions, that the mind possibly hesitates amid an obscure sense of many alternatives that cannot blend together. But limit the idea to, say, a landau, and the mental association declares itself more quickly. Say a landau coming down the street to opposite the door, and an image of many blended landaus that have done so, forms itself without the least hesitation.

Next, I found that my list of 75 words gone over 4 times, had given rise to 505 ideas and 13 cases of puzzle, in which nothing sufficiently definite to note occurred within the brief maximum period of about 4 seconds, that I allowed myself to any single trial. Of these 505, only 289 were different. The precise proportions in which the 505 were distributed in quadruplets, triplets, doublets or singles, is shown in the uppermost lines of Table I. The same facts are given under another form in the lower lines of the table, which show how the 289 different ideas were distributed in cases of fourfold, treble, double, or single occurrences.

TABLE I.  
RECURRENT ASSOCIATIONS.

Total number of Associations.	Occurring in			
	quadruplets.	triplets.	doublets.	singles.
505	116	108	114	167
per cent. . 100	23	21	23	33
Total number of different Associations.	Occurring			
	four times.	three times.	twice.	once.
289	29	36	57	167
per cent. . 100	10	12	20	58

I was fully prepared to find much iteration in my ideas, but had little expected that out of every hundred words twenty-three would give rise to exactly the same association in every one of the four trials; twenty-one, to the same association in three out of the four, and so on, the experiments having been purposely conducted under very different conditions of time and local circumstances. This shows much less variety in the mental stock of ideas than I had expected, and makes us feel that the roadways of our minds are worn into very deep ruts. I conclude from the proved number of faint and barely conscious thoughts, and from the proved iteration of them, that the mind is perpetually travelling over familiar ways without our memory retaining any impression of its excursions. Its footsteps are so light and fleeting, that it is only by such experiments as I have described that we can learn anything about them. It is apparently always engaged in mumbling over its old stores, and if any one of these is wholly neglected for a while, it is apt to be forgotten, perhaps irrecoverably. It is by no means keen interest and attention when first observing an object, that fixes it in the recollection. We pore over the pages of a 'Bradshaw,' and study the trains for some particular journey with the greatest interest; but the event passes by, and the hours and other facts which we once so eagerly considered become absolutely forgotten. So in games of whist, and in a large number of similar instances. As I understand it, the subject must have a continued living interest in order to retain an abiding-place in the memory. The mind must refer to it frequently, but whether it does so consciously or unconsciously, is not perhaps a matter of much importance. Otherwise, as a general rule, the recollection sinks, and appears to be utterly drowned in the waters of Lethe.

The instances, according to my personal experience, are very rare, and even those are not very satisfactory, in which some event recalls a memory that had lain *absolutely* dormant for many years. In this very series of experiments, a recollection which I thought had entirely lapsed appeared under no less than three different aspects on different occasions. It was this: when I was a boy, my father, who was anxious that I should learn something of physical science, which was then never

taught at school, arranged with the owner of a large chemist's shop to let me dabble at chemistry for a few days in his laboratory. I had not thought of this fact, so far as I was aware, for many years; but in scrutinising the fleeting associations called up by the various words, I traced two mental visual images (an alembic and a particular arrangement of tables and light), and one mental sense of smell (chlorine gas) to that very laboratory. I recognised that these images appeared familiar to me, but I had not thought of their origin. No doubt if some strange conjunction of circumstances had suddenly recalled those three associations at the same time, with perhaps two or three other collateral matters which may still be living in my memory, but which I do not as yet identify, a mental perception of startling vividness would be the result, and I should have falsely imagined that it had supernaturally, as it were, started into life from an entire oblivion extending over many years. Probably many persons would have registered such a case as evidence that things once perceived can never wholly vanish from the recollection, but that in the hour of death, or under some excitement, every event of a past life may reappear. To this view I entirely dissent. Forgetfulness appears absolute in the vast majority of cases, and our supposed recollections of a past life are, I believe, no more than that of a large number of episodes in it, to be reckoned in hundreds or thousands, certainly not in tens of hundreds of thousands, which have escaped oblivion. Every one of the fleeting, half-conscious thoughts which were the subject of my experiments admitted of being vivified by keen attention, or by some appropriate association; but I strongly suspect that ideas which have long since ceased to fleet through the brain, owing to the absence of current associations to call them up, disappear wholly. A comparison of old memories with a newly-met friend of one's boyhood, about the events we then witnessed together, shows how much we had each of us forgotten. Our recollections do not tally. Actors and incidents that seem to have been of primary importance in those events to the one, have been utterly forgotten by the other. The recollection of our earlier years are, in truth, very scanty, as any one will find who tries to enumerate them.



My associated ideas were for the most part due to my own unshared experiences, and the list of them would necessarily differ widely from that which another person would draw up who might repeat my experiments. Therefore one sees clearly, and I may say, one can see *measurably*, how impossible it is in a general way for two grown-up persons to lay their minds side by side together in perfect accord. The same sentence cannot produce precisely the same effect on both, and the first quick impressions that any given word in it may convey, will differ widely in the two minds.

I took pains to determine as far as feasible the dates of my life at which each of the associated ideas was first attached to the word. There were 124 cases in which identification was satisfactory, and they were distributed as in Table II.

TABLE II.

RELATIVE NUMBER OF ASSOCIATIONS FORMED AT DIFFERENT PERIODS OF LIFE.

Total number of different Associations.		Occurring								Whose first formation was in
		four times.		three times.		twice.		once.		
	per cent.		per cent.		per cent.		per cent.		per cent.	
48	39	12	10	11	9	9	7	16	13	boyhood and youth,
57	46	10	8	8	7	6	5	33	26	subsequent manhood,
19	15	—	—	4	3	1	1	14	11	quite recent events.
124	100	22	18	23	19	16	13	63	50	Totals.

It will be seen from the table that out of the 48 earliest associations no less than 12, or one quarter of them occurred in each of the four trials; of the 57 associations first formed in manhood, 10, or about one-sixth of them had a similar recurrence, but as to the 19 other associations first formed in quite recent times, not one of them occurred in the whole of the four trials. Hence we may see the greater fixity of the earlier associations, and might measurably determine the decrease of fixity as the date of their first formation becomes less remote.

The largeness of the number 33 in the fourth column, which

disconcerts the run of the series, is wholly due to a visual memory of places seen in manhood. I will not speak about this now, as I shall have to refer to it further on. Neglecting, for the moment, this unique class of occurrences, it will be seen that one-half of the associations date from the period of life before leaving college; and it may easily be imagined that many of these refer to common events in an English education. Nay further, on looking through the list of all the associations it was easy to see how they are pervaded by purely English ideas, and especially such as are prevalent in that stratum of English society in which I was born and bred, and have subsequently lived. In illustration of this, I may mention an anecdote of a matter which greatly impressed me at the time. I was staying in a country house with a very pleasant party of young and old, including persons whose education and versatility were certainly not below the social average. One evening we played at a round game, which consisted in each of us drawing as absurd a scrawl as he or she could, representing some historical event; the pictures were then shuffled and passed successively from hand to hand, every one writing down independently their interpretation of the picture, as to what the historical event was that the artist intended to depict by the scrawl. I was astonished at the sameness of our ideas. Cases like Canute and the waves, the Babes in the Tower, and the like, were drawn by two and even three persons at the same time, quite independently of one another, showing how narrowly we are bound by the fetters of our early education. If the figures in the above table may be accepted as fairly correct for the world generally, it shows, still in a measurable degree, the large effect of early education in fixing our associations. It will of course be understood that I make no absurd profession of being able by these very few experiments to lay down statistical constants of universal application, but that my principal object is to show that a large class of mental phenomena, that have hitherto been too vague to lay hold of, admit of being caught by the firm grip of genuine statistical inquiry.

The results that I have thus far given are hotch-potch results. It is necessary to sort the materials somewhat, before saying more about them.

After several trials, I found that the associated ideas admitted of being divided into three main groups. First there is the imagined sound of words, as in verbal quotations or names of persons. This was frequently a mere parrot-like memory which acted instantaneously and in a meaningless way, just as a machine might act. In the next group there was every other kind of sense-imagery; the chime of imagined bells, the shiver of remembered cold, the scent of some particular locality, and, much more frequently than all the rest put together, visual imagery. The last of the three groups contains what I will venture, for want of a better name, to call "histrionic" representations. It includes those cases where I either act a part in imagination, or see in imagination a part acted, or, most commonly by far, where I am both spectator and all the actors at once, in an imaginary mental theatre. Thus I feel a nascent sense of some muscular action while I simultaneously witness a puppet of my brain—a part of myself—perform that action, and I assume a mental attitude appropriate to the occasion. This, in my case, is a very frequent way of generalising, indeed I rarely feel that I have secure hold of a general idea until I have translated it somehow into this form. Thus the word "abasement" presented itself to me, in one of my experiments, by my mentally placing myself in a pantomimic attitude of humiliation with half-closed eyes, bowed back, and uplifted palms, while at the same time I was aware of myself as of a mental puppet, in that position. This same word will serve to illustrate the other groups also. It so happened in connection with "abasement" that the word "David" or "King David" occurred to me on one occasion in each of three out of the four trials; also that an accidental misreading, or perhaps the merely punning association of the words "a basement," brought up on all four occasions the image of the foundations of a house that the builders had begun upon.

So much for the character of the association; next as to that of the words. I found, after the experiments were over, that the words were divisible into three distinct groups. The first contained "abbey," "aborigines," "abyss," and others that admitted of being presented under some mental image.

The second group contained "abasement," "abhorrence," "ablution," &c., which admitted excellently of histrionic representation. The third group contained the more abstract words, such as "afternoon," "ability," "abnormal," which were variously and imperfectly dealt with by my mind. I give the results in the upper part of Table III., and, in order to save trouble, I have reduced them to percentages in the lower lines of the table.

TABLE III.

COMPARISON BETWEEN THE QUALITY OF THE WORDS AND THAT OF THE IDEAS  
IN IMMEDIATE ASSOCIATION WITH THEM.

Number of words in each series.		Sense imagery.	Histrionic.	Purely names of persons.	Verbal phrases and quotations.	Total.
26	"Abbey" series	46	12	32	17	107
20	"Abasement" „	25	26	11	17	79
29	"Afternoon" „	23	27	16	38	104
75						290
	"Abbey" series	43	11	30	16	100
	"Abasement" „	32	33	13	22	100
	"Afternoon" „	22	25	16	37	100

We see from this that the associations of the "abbey" series are nearly half of them in sense imagery, and these were almost always visual. The names of persons also more frequently occurred in this series than in any other. It will be recollected that in Table II. I drew attention to the exceptionally large number, 33, in the last column. It was perhaps 20 in excess of what would have been expected from the general run of the other figures. This was wholly due to visual imagery of scenes with which I was first acquainted after reaching manhood, and shows, I think, that the scenes of childhood and youth, though vividly impressed on the memory, are by no means numerous, and may be quite thrown into the background by the abundance of after experiences; but this, as we have seen, is not the case with

the other forms of association. Verbal memories of old date, such as Biblical scraps, family expressions, bits of poetry, and the like, are very numerous, and rise to the thoughts so quickly, whenever anything suggests them, that they commonly outstrip all competitors. Associations connected with the "abasement" series are strongly characterised by histrionic ideas, and by sense-imagery, which to a great degree merges into a histrionic character. Thus the word "abhorrence" suggested to me, on three out of the four trials, an image of the attitude of Martha in the famous picture of the raising of Lazarus by Sebastian del Piombo in the National Gallery. She stands with averted head, doubly sheltering her face by her hands from even a sidelong view of the opened grave. Now I could not be sure how far I saw the picture as such, in my mental view, or how far I had thrown my own personality into the picture and was acting it as actors might act a mystery play, by the puppets of my own brain, that were parts of myself. As a matter of fact, I entered it under the heading of sense-imagery, but it might very properly have gone to swell the number of the histrionic entries.

The "afternoon" series suggested a great preponderance of mere catch-words, showing how slowly I was able to realise the meaning of abstractions; the phrases intruded themselves before the thoughts became defined. It occasionally occurred that I puzzled wholly over a word, and made no entry at all; in thirteen cases either this happened, or else after one idea had occurred the second was too confused and obscure to admit of record, and mention of it had to be omitted in the foregoing table. These entries have forcibly shown to me the great imperfection in my generalising powers; and I am sure that most persons would find the same if they made similar trials. Nothing is a surer sign of high intellectual capacity than the power of quickly seizing and easily manipulating ideas of a very abstract nature. Commonly we grasp them very imperfectly, and hold on to their skirts with great difficulty.

In comparing the order in which the ideas presented themselves, I find that a decided precedence is assumed by the Histrionic ideas, wherever they occur; that Verbal associations

occur first and with great quickness on many occasions, but on the whole that they are only a little more likely to occur first than second; and that Imagery is decidedly more likely to be the second, than the first, of the associations called up by a word. In short, gesture-language appeals the most quickly to our feelings.

It would be very instructive to print the actual records at length, made by many experimenters, if the records could be clubbed together and thrown into a statistical form; but it would be too absurd to print one's own singly. They lay bare the foundations of a man's thoughts with curious distinctness, and exhibit his mental anatomy with more vividness and truth than he would probably care to publish to the world.

It remains to summarise what has been said in the foregoing memoir. I have desired to show how whole strata of mental operations that have lapsed out of ordinary consciousness, admit of being dragged into light, recorded and treated statistically, and how the obscurity that attends the initial steps of our thoughts can thus be pierced and dissipated. I then showed measurably the rate at which associations sprung up, their character, the date of their first formation, their tendency to recurrence, and their relative precedence. Also I gave an instance showing how the phenomenon of a long-forgotten scene, suddenly starting into consciousness, admitted in many cases of being explained. Perhaps the strongest of the impressions left by these experiments regards the multifariousness of the work done by the mind in a state of half-unconsciousness, and the valid reason they afford for believing in the existence of still deeper strata of mental operations, sunk wholly below the level of consciousness, which may account for such mental phenomena as cannot otherwise be explained. We gain an insight by these experiments into the marvellous number and nimbleness of our mental associations, and we also learn that they are very far indeed from being infinite in their variety. We find that our working stock of ideas is narrowly limited, but that the mind continually recurs to them in conducting its operations, therefore its tracks necessarily become more defined and its flexibility diminished as age advances.

# ON THE ELECTRICAL EXCITABILITY OF THE SKIN.

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and

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THE object of the following pages is threefold. First, to indicate the conditions which any rational method must fulfil which pretends to give us accurate results as to the electrical excitability of the cutaneous nerves; these conditions will be found to depend partly upon the behaviour of the electrical current, partly upon certain anatomical peculiarities of the skin. Secondly, to criticise in the light of the principles set forth the chief methods hitherto proposed to estimate the electrical sensibility. Third, to propose a new method more simple and rational; and state some of the results already obtained from its application.

## I.

According to the general law of excitation of nerves, formulated by Du Bois-Reymond, this excitation depends upon the *rapidity* with which the electrical density changes in the nerve, and not with the *absolute value* of that change.

Hence we ought, for measuring the electrical excitability of a nerve, to determine the rapidity of change in the density necessary to excite it; and this is what Dr. v. Fleischl's<sup>1</sup> ingenious instrument, the Rheonome, enables us to do. Only

<sup>1</sup> "Untersuchung über die Gesetze der Nervenenerregung," III. Abhandlung. Stzber. d. k. Academie der Wissensch. zu Wien. Vol. lxxvi., Part III., p. 138.

such measurements are much too complex to be of any practical clinical value. It is, however, possible to attain the same object by simpler means, through the elimination of some of the variables in the experiment. Thus if the current is made through the nerve with always the same rapidity, so that it reaches its maximum intensity in the same space of time, we may measure the excitability of the nerve by the density of the current flowing through it. Or again, if the diameter of the nerve remains constant, its excitability may be estimated by the strength (intensity) of the current.

It is easy to base upon these considerations a rational method for measuring the electrical excitability of the skin. This would consist in giving the galvanometric value of the currents necessary to produce the minimum sensation in every part of the skin; provided that these two conditions are observed: (1) the make and break of the current must be made every time with the same rapidity; (2) the surface of contact of the electrode with the skin must always be the same. It is evident that by using this method we are independent of all the variations in the resistance, permanent or incidental, originating in the parts to be examined. Let it be clearly understood, however, that we assume here a direct galvanometric measurement of the current strength, and not an estimation of it by resistances introduced either in the circuit itself, or in a derived current.<sup>1</sup>

Given two points of the skin of different electrical resistance; we must, to obtain the same current-strength in both cases, use a different number of elements. Once the same current-strength obtained, the excitation is the same in both cases, provided always the other conditions be fulfilled, i.e. equal rapidity of make and break, and equal extent of excited surface. Now it is clear that under such circumstances, if the excitability be the same, the effect of the excitation must be the same also.

By these means, then, we are made independent of the source of error pointed out frequently before and arising from

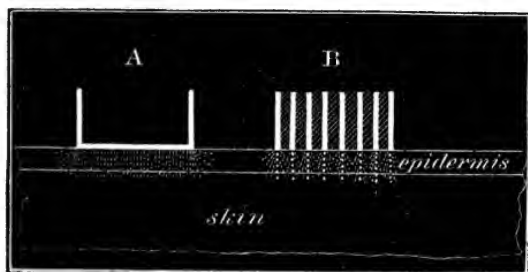
<sup>1</sup> The latter process, so often employed by electrotherapeutists, after the example of Brenner, ought to be entirely abandoned. Applied in researches such as those we have described, for instance, it would give entirely erroneous results.



the variable thickness of the epidermis. Yet, in order to be able to measure the excitability, not of the collective nervous elements of the skin, but of the cutaneous nerves themselves, we must be able to eliminate the sources of variation arising from the structural differences of the nervous supply in different parts of the skin.

We have, it is true, no direct anatomical demonstration of such differences; but physiological observations, such as the measurements with E. H. Weber's compasses, allow us to suppose that the number of nervous endings in a unit of cutaneous surface varies in different parts of the body. If this supposition be true, it necessarily follows that an electrode of the same surface applied to different parts of the body ought to produce excitations of various strengths merely on account of the variable richness of the skin in nerve-endings.

The way to eliminate this source of error consists in always exciting the skin at the same number of points by means of a special form of electrode. The simplest would be a pointed electrode which would eliminate the influence of a variable number of excitations by reducing this number to a minimum; but its sharpness would make an accurate estimation of the minimal electrical excitation difficult. The following form of electrode, however, satisfies the present condition sufficiently well: It consists in a small cylindrical bundle of well insulated wires; and it is not difficult to understand the important difference between the effects of such an electrode and those of one in the shape of a solid cylinder of the same diameter.



The figure makes this difference evident. The excitation produced by B will be stronger than that produced by A

because the surface of the metallic contact with the skin is less, and hence the density of the current greater. Again, and this is the important point, the number of the excitations in the case of B will be more uniformly the same, whilst in the case of A it will depend upon the abundance of cutaneous nerve-endings. For it may be assumed that, with B, none but the shortest currents from the wires through the epidermis will stimulate the nerves; that is, none but the nerve-fibres lying nearest to the wires (the points of greatest electrical density) will be stimulated—especially as we here deal merely with minimal excitations. We shall see further on that experience has completely borne out these theoretical considerations.

There is yet a condition that has not hitherto been attended to in testing the sensibility of the skin, and which must however, have some influence on the results of the experiments. One of us (Dr. Tschiriew) in a paper forthcoming in the *Archives de Physiologie* (Charcot, Vulpian, and Brown-Séquard) starting from physiological and pathological data, shows that it is necessary to assume the interruption of all the centripetal paths in the grey matter of the spinal cord; and that the differences in the duration and intensity of the excitation necessary to produce the same effect may be explained by the variations occurring in that interruption among the several afferent nerves. Hence it follows that, in order to eliminate differences due to any such variations possible among the afferent cutaneous nerves, it is necessary to allow a certain duration to each excitation.

In order to fulfil this condition if the continuous current were used, and also to avoid the mechanical and electrotonic influences of this current upon the tissues, we should require an apparatus which would interrupt the current, and at the same time reverse it each time, at a uniform rate of speed. This obviously complicates the process so much as to make it inapplicable for clinical observation. But before we pass to the description of a simpler and more practical method we must say a few words on those hitherto proposed, and examine how far they fulfil the conditions just laid down.

## II.

In 1864 Munk and Leyden ('Untersuchungen über die Sensibilität im gesunden und kranken Zustande,' Virchow's 'Archiv,' vol. xxxi. page 1) described the following method for testing the electro-cutaneous sensibility. A pair of wooden compasses were fitted with metallic terminations connected with the secondary coil of a Du Bois-Reymond's induction apparatus. The points were fixed at a constant distance from one another (1 cm.), and applied to the skin. The sensibility was then determined by noting the distance between the two coils when the point of minimum sensation was reached. Simple make or break induced currents, of constantly the same rapidity, were used. This method led its authors to the following results: (1) Different regions of the skin differ in their absolute sensibility in much the same degree as in their sense of space (Weber).<sup>1</sup> (2) The electro-cutaneous sensibility decreases in the following order: face, trunk, upper arm and thigh, elbow and knee, tips of fingers and toes.

The authors themselves define the value of these measurements by adding that the local thickness of the epidermis, as well as the local abundance of nerve fibres, must influence the results obtained. This method is indeed faultless if our object is merely to determine the electro-sensibility of the skin as a whole, with all its incidental peculiarities, such as the varying thickness of its epidermis, abundance of its sensitive elements, conductivity of its subjacent structures, and mode of interruption of its afferent nerves in the spinal cord. For it is admissible that in every healthy individual the *distribution* of these anatomical peculiarities of the skin in its different parts follows the same law. On the other hand it would be impossible to draw any conclusions whatever from results obtained by this method about the sensibility of the cutaneous nervous elements themselves, since these results have been obviously modified by the various conditions just enumerated.

<sup>1</sup> This hardly agrees with the results obtained by the authors themselves: the tips of the fingers, for instance, are more developed for the sense of space than the trunk.

Bernhardt ('Electrotherapeutische Notizen,' *Deutsches Archiv für Klinische Medizin*, 1877, vol. xix. p. 382), whilst objecting to the foregoing, the practical difficulties in the comparison of results obtained with different induction apparatus, proposes the following method: to measure the electrocutaneous sensibility by the resistances which must be intercalated in a derived circuit in order to reach the minimum sense of pain. The "modus operandi" is as follows: The patient holds the positive electrode of a battery of thirty Stöhrer's elements in his left hand. The sensibility is tested by means of a wire brush connected with the negative pole. A derived current is established through a rheostat, and the resistances read off as soon as pain is experienced. These readings give the measure of the sensibility.

This method offers no advantage over that of Munk-Leyden; the results obtained even for the same individual are hardly comparable among themselves. The relations between the variations in the rheostat resistances and the current strengths thus set up through the body, are obviously far more complex than those between the distances of the coils and the strength of the induced currents in a non-graduated apparatus. Besides, given the number of cells included in the inducing circuit, and the number of turns of wire in both coils, the true value of the distances between the two coils noted in any experiment can at any time be ascertained; whereas the value of the rheostat resistances could be determined only by the repetition of all the measurements upon the same person, and by a process the complexity of which is in itself greater than the graduation of an induction apparatus.

Again the determination of the minimum sensation obtained on exciting by a single make or break of a continuous current, owing either to the longer time it takes to attain its maximum intensity or to the electrotonic influences it exerts upon the tissues, is much less precise than that produced by the induced current. This is easy to demonstrate upon oneself. The matter becomes of still greater importance when we have to do with patients whose intelligence is not always of the keenest. We may also recall the well-known phenomenon explainable by the electrotonic effects of the galvanic current:

that, when we think we have reached the minimum current strength that will produce sensation, we find, on controlling the experiment by further diminishing the current, sensation will now manifest itself to much weaker stimulations than at first.

But the greatest objection to Bernhardt's method is the fact that the resistance of the body included in the circuit varies with every new position of the negative electrode—a source of error which he gives us no means to remedy. As an instance of the results he obtains we may quote the following measurements: for the tip of the nose, from 50 to 60 Siemens' units,<sup>1</sup> intercalated resistance sufficed; whereas 2000 to 3000 S. U. were necessary for the tips of the fingers and toes! As to the general conclusions reached by the author, they agree with those of Munk and Leyden. He, too, refrains from drawing any as to the irritability of the cutaneous nerves themselves.

The current number of the 'Archiv für Psychiatrie' contains a paper by Drosdoff ('Untersuchungen über die elektrische Reizbarkeit der Haut bei Gesunden und Kranken,' vol. ix. part 2). The highly elaborate form in which these researches are presented, compels us to pay more attention to them than their intrinsic value deserves. Some of the objections raised by the writer to the previous methods are either utterly groundless or have been anticipated by their authors themselves. Thus, for instance, when he objects that the results obtained by Munk and Leyden are vitiated by the varying abundance of nerve elements in the skin, he forgets that, so far from falling into such an error, these authors had pointed out this fact as forbidding them to conclude from the sensibility of "the skin" to that of "the cutaneous nerves." This does not prevent Dr. Drosdoff from falling into it himself, though judged from his own standpoint, the method he uses is still more defective than that of the above-named authors. Like Bernhardt he fixes one of the electrodes on some part of the body (the sternum), and explores the sensibility by means of the other electrode in the shape of a wire brush. He uses a non-graduated induction apparatus, and estimates the sensibility by the distance

<sup>1</sup> Siemens' unit of resistance is equal to .97 ohm nearly.

between the two coils, like Munk and Leyden. This method is thus merely a combination of the two former ones, from the imperfections of which it does not escape. It unites the limited applicability of the first (Munk-Leyden) to the weakest point of the second (Bernhardt), in that it introduces a different resistance into the circuit, with every change of position of the exciting electrode. Neither do the results obtained by Drosdoff differ materially from those of the previous observers. In order, however, to find out whether the differences in the sensibility discovered in different parts of the body are not simply due to differences in the resistance of these parts, the author has made numerous measurements of what he calls "epidermic resistances." For this purpose he fixes one of the electrodes of a battery of twelve Stöhrer's elements on the sternum, and applies the other to the various parts of the body, the sensibility of which has been tested. A galvanometer with 150 turns of wire is introduced in the circuit, by the readings of which the resistances are estimated. Now it is evident that it was the resistance of the whole portion of the body included between the electrodes and not that of the epidermis that the author was measuring. Again, if the instrument used was a simple sinus-galvanometer, and was not graduated in absolute units—and he says not a word on this important point—it is evident that his measurements did not correspond to the absolute value of the electrical forces. As it is, one is struck at first sight by the parallelism between the series of numbers obtained by the resistance-measurements and those obtained by the sensibility-measurements at different points of the body; but, strange to say, the author draws from them the following conclusion: that there is no relation between the differences of resistance and of sensibility of the same points. Let the reader judge for himself. At page 213, under the title of "electrical zones," we find the following numbers:

M.S. . .	232·5	212·0	200·7	193·3	154·2	188·1
M.P. . .	165·2	156·5	146·4	142·3	123·6	142·3
Dev. . .	22·2°	9·3°	6·9°	4·5°	2·5°	6·5°

And at page 215:

M.S. . .	184·6	184·2	178·1	138·8		
M.P. . .	143·0	140·0	133·0	117·6		
Dev. . .	7·1°	5·3°	3·4°	2·9°		

M.S. stands for minimum sensation; M.P. for minimum pain; Dev. for deviation in degrees of the needle. Each vertical column of numbers corresponds to one of the author's zones. These numbers are means of ten observations, and therefore have a higher intrinsic value than each of their component factors. The parallelism which we observe among them is quite as marked as what we could expect from the imperfect method used and the peculiarities of human bodies.

The way Dr. Drosdoff escapes the conclusions forced upon him by his own data is to pick out among the individual factors a few exceptional numbers in which the parallelism does not exist; and from these exceptions, due probably to unavoidable errors of observation, he concludes that there is no such thing as a relation between the variations of the resistance and of the sensibility. Further, upon this extraordinary conclusion, the author bases another assertion of still higher import, and which he gives as the grand result of his investigation, viz.: "that the differences of sensibility at different points of the skin are due to differences in the excitability of the nerves themselves." We shall state evidence further on to disprove the author's conclusions, from the results of our own experiments. They are far from being deducible from his own measurements, and indeed it would seem that he has conspicuously brought into evidence the great influence variations of resistance have exerted upon the results obtained. Elsewhere he is led astray by his ignorance of the properties of the instruments he used. He says (page 219), "The difference ('Verhältniss') between the minima of sensation and those of pain varies between 6 and 88 mm. (mean: 11-73.6) distance between the two coils. These distances diminish, in the case of painful impressions, with the increase of current strength." This statement is false if by it is meant that there

is any relation between the two kinds of sensation; for the author's results have been obtained with a non-graduated apparatus, and is ascribable to the mere absence of proportionality between the distances of the coils and the strengths of the corresponding currents.

### III.

From what we have said, it is clear that the method of Munk-Leyden is sufficient for measuring the sensibility of the skin; and that none of the methods hitherto proposed enables us to test the electrical excitability, absolute or relative, of the cutaneous nerves themselves.

We have pointed out previously a rational method for accomplishing this object. But, as we saw, though correct, it is too complex to be applicable to clinical purposes. We have, however, another one to propose, which is simple, though fulfilling all the conditions which have been laid down previously. We now pass to the description of this method, and of the results we have been able to obtain by its application.

Its principles are the following: (1) Elimination of all the sources of variation in the strength of the currents due to the variable thickness of the epidermis,<sup>1</sup> and the different positions of the electrodes, &c., by intercalating in the circuit such resistances as to make such variations insignificant; (2) Elimination of the influence of the variable abundance of nervous elements in the skin by exciting it at a constant number of points disposed over a constant surface. The latter is effected by using the form of electrode described previously, and composed of a solid cylinder of insulated wires.

In order to fulfil the first condition, it was necessary to know what resistances the human body could offer in such experiments. The measurement of these resistances is far from being such a simple process as is commonly thought. The conditions of experimentation from which they are ob-

<sup>1</sup> This condition has often been held up as very unfavourable to the appreciation of the sensibility of the cutaneous nerves, and it is thought that were that thickness known at various parts of the body much could be gained thereby. We hold the opposite view; for if we cannot eliminate by the method itself the influence of the epidermis, how can we hope to be able to do so by calculation when we are acquainted, ever so precisely, with its thickness at every part of the body?



tainable are so various as to make it impossible to speak absolutely of the electrical resistance of a part of the body, but only of its resistance under this, that, or the other condition. For instance, according to the details of the process of experimentation adopted, the "resistance of the forearm" may vary from about two to forty (or more) thousand ohms. The main circumstances influencing the results are: (1) the size, shape, and moisture of the electrodes; (2) the pressure with which they are applied; (3) the strength of the currents employed; (4) the condition of the parts tested, e.g. the degree moisture of the epidermis, the previous application of a more or less strong current, &c.

The first circumstance is of easy explanation. The fact that resistance diminishes with the pressure exerted upon the electrode is not so satisfactorily accounted for. It is more readily observed in parts of the skin with a compressible, rather than a hard, subjacent tissue. The electrical resistance may be diminished, on increasing the pressure, by one-half, two-thirds, or even more.

The variations dependent upon the current strength are illustrated by the well-known fact that when first applied to the skin a current from a large number of elements may be weak, but soon becomes stronger, showing that the patient's resistance diminishes. This diminution must be, partially at least, explained by the cataphorical effect of the current (du Bois-Reymond) by which liquids are conveyed into the integuments either from the electrode or from the subjacent tissues. Hence weak currents must be used for measurements in order to eliminate this source of error. The fourth condition referred to above may also be reduced to the increased moisture of the integuments. The following experiment will illustrate it. An electrode is fixed to the back, and another applied to the dorsal surface of the forearm. The resistance is found to be about 10,000 ohms. After a little rubbing of the arm with the electrode, and a strong current, the resistance is found to have sunk below 3000 ohms. The practical outcome of all this for measurements of body-resistances is that, first, all the conditions of observation must remain the same throughout; second, all these conditions must be carefully described in

every case. If these requisites are not fulfilled, the data obtained cannot have the slightest value.

Our measurements were taken as follows: A metallic plate,  $5 \times 12$  cm., covered with wet wash-leather, was fixed to the upper part of the back; the various parts of the body were explored with a metallic disk 7 mm. in diameter, also enclosed in wet wash-leather. The latter electrode was fixed to an Eulenburg's baræsthesiometer, in order to ensure the same amount of pressure everywhere. The pressure used was of 150 grammes. The skin at the point of application was moistened, and the time taken for each observation restricted to a few seconds, in order to avoid differences arising to the unequal imbibition of the epidermis. Several observations were taken for each region, but the electrode was never re-applied to exactly the same spot, or only so after some time had elapsed. The battery used was a Gaiffe-Leclanché, and the number of elements used (4-14) chosen so as to obtain at every part a weak current (.2 to .5 milliveber),<sup>1</sup> and to avoid the disturbing effect of strong currents on the tissues. The following table gives our own resistances in ohms:

	Ohms.	Ohms.
Tip of nose . . . . .	12,000	8,100
Forehead . . . . .	4,000	3,000
Cheek . . . . .	8,200	6,400
Forearm (post) . . . . .	18,750	14,000
„ (anter.) . . . . .	20,000	..
Hand (dorsal) . . . . .	21,000	15,500
„ (palmar) . . . . .	42,000	48,000
Tips of fingers . . . . .	65,000	46,000
Leg . . . . .	21,000	23,000
Foot (dorsum) . . . . .	22,000	24,600
„ (sole) . . . . .	80,000	50,000
Tips of toes . . . . .	60,000	60,000

<sup>1</sup> A current of one milliveber is that obtained with one volt through one thousand ohms. The electromotive force of a Daniell's cell is nearly one volt.

These numbers hold only for limited areas of the regions investigated. The different portions of the leg, for instance, tested under the same conditions throughout will vary in resistance according to the peculiarities of the epidermis and subjacent tissues. Similar measurements made with the electrode used in testing the sensibility (a cylindrical fasciculus of insulated wires) gave still more considerable differences, especially with the dry skin. In this way the resistances of the tips of the fingers and toes may amount to 100,000 ohms and more.

Hence we see that in order fully to eliminate the influence of the body-resistance in investigating the sensibility on Bernhardt's principle, at least a 2-million ohms' resistance is to be introduced in the circuit; and it is easy to perceive how much this author's and Drosdoff's results must have been influenced by these sources of fallacy which they have ignored.

For the investigation of the sensibility, our method consisted in fixing a large neutral plate-electrode on the back; and in using as differential or exciting electrode a metallic wire brush, of which the wires were insulated with sealing-wax, and brought together to form a cylindrical bundle of 75 mm. diameter. Care was taken that the exciting surface was as smooth as possible. This electrode was mounted on an interrupting handle. A Du Bois-Reymond's induction apparatus, fed by two Bunsen's cells, was used. The secondary coil (600 metres of 0.225 mm. copper wire) was used, and in the circuit included a resistance of upwards of three million ohms, formed by a slip of vulcanite overlaid with a film of plumbago. The hammer of the apparatus was made to vibrate at a rapid rate, and the current closed by means of the key through the previously applied electrode. This is a most important precaution, as "dabbing" the skin with the current on would produce such variations in the density of the current as to vitiate all the results. The minimum point of sensation was sought by adapting the distance between the two coils, whilst excitations were made by making and breaking the current by means of the key, the electrode remaining immoveable *in situ*. Finally, sufficient time was allowed for each excitation.

In this way we have arrived at very unexpected results.

In opposition to what has been stated by previous observers, we have found that *the electrical excitability of the skin, or rather of the nerves of the skin, is the same at every part of the body.* There always occur slight differences (amounting to from a few mm. to 1 cm. of coil); but on the one hand these differences are not always in the same sense, on the other their absolute value is too small<sup>1</sup> to claim any special signification.

Several points must be attended to in order to obtain reliable data with our method. (1) The elements feeding the coil must be *constant* (this excludes Leclanché's, and all single fluid cells, such as Stöhrer's, Smee's, &c.). We used two Bunsen's. (2) The conducting wires must be thoroughly insulated from every surrounding object, owing to the enormous "tension"<sup>2</sup> of the current. (3) As before mentioned, the excitation must begin after the electrode is *in situ*. (4) The portion of skin tested must be moistened. At first sight this last condition appears superfluous; for how, it is asked, can the hygrometric state of the skin have any influence when the circuit contains already such an enormous resistance?

We venture to offer the following explanation of this phenomenon. It is not due to any change in the resistance of the skin; because such a change would, as objected, be of no appreciable influence under the circumstances, and because also—a fact opposed to such a supposition—dry skin is more excitable than moist skin. But, as it has been experimentally shown (Tschiriew, 'Ueber die Nerven- und Muskelerregbarkeit,' in Du Bois-Reymond's Archiv f. Anat. & Phys., 1878, p. 494), electricity distributes itself, in a transverse section of every conductor it passes through, always in an inverse ratio to the resistance (Kirchoff's law), *independently of any resistance in the circuit.*

<sup>1</sup> Though the curve representing the relation between the distances of the two coils and the current-strengths corresponding to these distances is entirely independent of the resistances in the circuit of the secondary coil, the absolute value of these currents is *not* independent of these resistances, which appear in the curve as one of the parameters. Since then, in our experiments where the circuit included an intercalated resistance of several million ohms, the variations of 1 cm. or less in the coil-distances can correspond but to very small variations in the current-strengths.

<sup>2</sup> De Watteville, "On the Nature of Electrical Tension."—'Medical Times and Gazette,' Sept. 1877, and 'Med. Electricity,' Chap. I.

Hence even in our experiments where several million ohms' resistance was included in the circuit, even in the case of the dry skin where we must assume differences of resistance between the dry epidermis and the sudoriparous ducts, the whole current would find its way through the points of least resistance. Hence a current, which on a moist skin would have been hardly felt, may become even painful on a dry one. It is a well-known fact that the excitation of the dry skin produces a pungent sensation; of the moist skin a milder sensation—the latter being more evenly diffused than the former. Again, this difference is less marked in some regions than in others; for instance, less at the finger-tips than on the cheek or dorsum of hand. Hence appears the importance of eliminating this source of fallacy.

Another capital precaution in sensibility-testing is to avoid placing the electrode on any nervous twig. The peculiar sensation evoked will tell us if it has been done; and as a rule we must always choose the least sensitive spots for exciting. Generally speaking it will be advisable to avoid those spots where the epidermis is very thick; this latter condition would involve not only a higher resistance, but also a greater thickness of the layer interposed between the nervous elements and the electrode, and thus increase the diffusion of the currents before they reach the nerve-endings. In order to illustrate the influence of the kind of electrode we use (cylindrical bundle of insulated wires) in eliminating the influence of the variable abundance of cutaneous nerve-elements, we have compared its effects with those of a solid cylinder of the same diameter (7 mm.). With the former, as above related, we found the excitability of every part of the body practically the same. With the latter, we found it different, and to a certain extent varying in the same ratio as Weber's sense of space. Thus:

	cm.		cm.
Nose . . . . .	7•	Hand (palm) . . . .	5•
Forehead . . . . .	7•2	Tips of fingers . . .	8•3
Lips . . . . .	7•3	Leg . . . . .	3•
„ (red part) . . . .	5•3	Foot (dorsum) . . .	4•
Forearm (front) . . .	6•6	„ (sole) . . . . .	0•
„ (back) . . . . .	5•2	Tips of toes . . . .	7•5
Hand (back) . . . .	5•5		

In the interpretation of these data, however, it must be noted that we used a non-graduated induction apparatus, fed by two Leclanché's, with several million ohms' resistance in the circuit. We have seen before that the absolute strength of induced currents diminishes in an inverse ratio to the intercalated resistance. Hence the absolute value of the excitations corresponding to the coil-distances just given is much less than if the body alone had been included in the circuit.

The different results obtained from the use of the two forms of electrodes can be explained only by the fact of the varying nervous supply of different regions of the skin, and the elimination of its influence in the case of the special electrode.

Another observation we have made in the course of our experiments and which illustrates the importance of the shape of the electrode in exciting the skin, is, that if the bundle of insulated wires was made into an elliptical instead of a cylindrical column, the results obtained depended upon the relative position of its long axis with reference to the prevailing direction of the subcutaneous nerves. If the long axis coincides with this direction (as for instance in the limbs in the direction of their length) the excitation produced is less powerful than when it is transverse to the general course of the nerves. The same result is observable when a solid elliptical electrode is used.

This fact does not disprove our assertion about the possibility of eliminating the influence of the relative abundance of nerve-elements in different parts of the skin by our electrode. For, besides the arguments derived from comparative experiments, it is hardly possible to conceive such a regular distribution of nerve-endings in the skin that a mere alteration in the direction of the electrode should be followed by a change in the number of the endings influenced. We must assume, then, that we have here to do with a new factor of influence in the measurement of sensibility, viz., the mode of division and subdivision of the subcutaneous nerves.

An analogous phenomenon is observed with Weber's compasses. If at the same spot the minimum distance of distinct

impressions is sought in various directions, it is found (especially in the limbs) that these distances are greater in a parallel than in a transverse direction to the main course of subcutaneous nerves. Hence Weber's "circles" are ellipsoids with their long diameter directed along those nerves.

The explanation of this phenomenon is, that when the long axis of the electrode coincides with the main direction of the nerve-trunks, the larger number of exciting points influence the terminations of the same system of ultimate fibres of some one nervous twig. If, on the other hand, the long axis of the electrode is directed transversely to the nerves, the number of excited points of each twig will be less, but the number of those twigs stimulated will be greater.

Hence it will be observed that for testing the sensibility, electrodes with a circular surface of contact are alone to be used in order to eliminate this disturbing factor from the results.

The advantages of the method we have adopted are, that it is simple, easily applicable in clinical investigations,<sup>1</sup> and that its results, to be understood, do not require any comparison with tables of the distribution of sensibility in different parts of the body. As an example of its application we subjoin the results obtained in a case of bulbar paralysis with lateral sclerosis. All the tendon-reflexes were exaggerated, especially on the right side. The sense of touch was nowhere greatly impaired, but there was decided diminution of the sensation to pain, on the right side especially.

<sup>1</sup> In order to facilitate the application of our method, we are endeavouring to produce an appropriate electrode. Our present model is to consist of a tube of non-conducting material containing in its upper part a rod of resisting substance—kaolin and graphite—of two million ohms; in its lower, a cylinder of metal, the extremity of which forms the exciting surface. This surface will be subdivided by a system of intersecting grooves filled with an insulating substance, into a number of exciting points, as shown in the diagram already figured. This little appliance, mounted upon an ordinary interrupting handle, will be all that is required, besides an induction apparatus with a moderately long and fine secondary wire and a constant element.

Part of Body.	Distances of Coils.	
	Right.	Left.
Nose . . . . .	6·	7·2
Forehead . . . . .	6·1	6·5
Cheek . . . . .	4·3	5·5
Back (lower dorsal) . . . . .	0·5	3·6
Arm . . . . .	4·	5·6
Forearm (back) . . . . .	3·5	5·3
„ (front) . . . . .	3·7	4·5
Hand (back) . . . . .	2·2	4·6
„ (palm) . . . . .	>0	0
Tips of fingers . . . . .	3·2	5·5
Leg . . . . .	0	5·
Foot (dorsum) . . . . .	0	3·7
„ . . . . .	>0	0
Tips of toes . . . . .	>0	>0

The same induction apparatus as previously was used, with two Bunsen cells. In the circuit a resistance of more than three million ohms was intercalated.



# THE EYE-SYMPTOMS IN LOCOMOTOR ATAXIA.

(A CLINICAL LECTURE.)

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GENTLEMEN,—I purpose to-day to explain and illustrate the Eye-symptoms which occur in Locomotor Ataxia. They are very common, as you will perceive when I tell you that they were present, in one form or other, in 41 out of 64 cases tabulated by Eulenburg,<sup>1</sup> and in 18 out of 20 cases of which I have notes. It is mainly upon a study of these 20 cases that the description I am to give you is founded.

There are four sets of symptoms :

- I. Double Vision, Squinting, and Ptosis.
- II. Changes in the Pupil.
- III. Amblyopia and Amaurosis.
- IV. Colour-Blindness.

## I. *Double Vision, Squinting, and Ptosis.*

These temporary paralyses are very common affections, and manifest themselves often by double vision without apparent squint, sometimes by double vision only when the eyes are turned in particular directions, often also by distinct squint and by ptosis. The existence of the last-named symptom forbids the adoption of any hypothesis of spasm or ataxia, and shows clearly that these conditions are manifestations of the well-recognised tendency to temporary paralysis which exists throughout the course of the disease, and especially in its early stages.

<sup>1</sup> 'Lehrbuch d. Nervenkr., 2te Aufl. Bd. ii. s. 448.

Out of the 64 cases recorded by Eulenburg, 25 had strabismus. Of these 25, 19 had divergent strabismus, the third nerve being paralysed; 6 had convergent strabismus, the sixth nerve being affected; and 4 of the divergent cases had, in addition, paralytic ptosis. Of my 20 cases, I find that 8 had paralytic eye-symptoms, 5 having strabismus, 3 ptosis, and 4 diplopia, without manifest squint. I have not seen dilatation of the pupil occur along with the squint in any of these cases, and from this, as well as the other facts, you observe that all the branches of the third nerve are not necessarily affected together.

In illustration of these affections I may recall the case of a man, who was under observation here, suffering from locomotor ataxia in an early stage, and who exhibited no ptosis nor distinct tendency to squint, though he complained of double vision whenever his eyes were directed much to one side, or when an object was held near the eyes. Here you observe that the branch to the levator palpebræ superioris was unaffected, and in ordinary positions of the eye there was no double vision; but when the patient looked very much to one side, the third nerve of the one eye failed to produce an action equal to that of the sixth nerve of the other, and thereby double vision was produced; and again, when the eyes were converged upon a near object, the paresis manifested itself in the same way. In the case of a man, Cowan, who was for many years under my observation, and whom some of you will remember, a similar set of symptoms occasionally existed. In 1873, for example, I find it noted that he was troubled with double vision, when he looked to one side. The same man's history affords good illustration of the more obvious conditions of ptosis and squinting.

Let me mention another case, that of a man named Garrow, who was under my care in 1874. He told us that his earliest symptom was a sudden attack of giddiness, with ptosis and squinting, which occurred in 1863; that he was for thirteen weeks under the care of Mr. Walker for this malady, and only at the end of that time got completely rid of it. It recurred in the following year, and again in 1866, each time without apparent cause. Patients sometimes say that they noticed

nothing wrong, until their friends drew attention to the drooping of the eyelid or the squint.

Now these symptoms are often premonitory of the commencement of the typical symptoms of the disease. These paralyses may, however, occur after the locomotor ataxia is well advanced, and I have known them to reappear with each attack of gastric crisis, and to linger for some time after it had passed away. Von Graefe has remarked that in these cases there is a striking diminution of the tendency to the blending of images, on which binocular vision depends; that is to say, that whereas in cases of slight double vision an effort is instinctively made by movements of the eye to bring the two images into one, in this condition the attempt is scarcely made. He suggests that this points to a central origin of the affection, which view is rendered more probable by the sudden onset and temporary character of the symptom, as well as by its occurrence in connection with gastric crises.

This group of symptoms, then, you will remember is a local manifestation of the general tendency to temporary paralyses.

## II. *Alterations of the Pupil.*

*a. Mydriasis.* This condition was observed by Eulenburg in 9 out of his 64 cases; in 3 the dilatation being double, in 4 single, and in 2 accompanied by myosis in the other eye. I have not noted this condition in any of my cases. Eulenburg states that it appears to occur mostly in the later stages of locomotor ataxia. Had it been an early symptom one might readily have explained its occurrence by one or other of two hypotheses, viz. either a paralysis of the third nerve, or an irritation of the cilio-spinal fibres. The latter is made the more likely, as Eulenburg suggests, by the circumstance that there is no defect of the power of accommodation, and that during accommodation the pupil acts, both of which facts indicate that the third nerve is unaffected. If, however, it be true that mydriasis occurs mainly in the later stages, it is difficult to understand how irritation of the cilio-spinal nerves should arise, unless in cases which have had no affection of the cilio-spinal region till a late period, and that then irritation has

preceded the paralysis. Here again is a difficulty, for were it the case that irritation always preceded paralysis, we should expect mydriasis to be a frequent early symptom of locomotor ataxia, seeing that myosis is common. Perhaps this is actually the case, and the condition is overlooked on account of the absence of concomitant symptoms.

β. Myosis has long been well known as a frequent eye-symptom in locomotor ataxia. It is referred to by Romberg, Remak, Duchenne and Trousseau, as well as by recent writers. Sometimes it is so marked as at once to attract the observer's attention. Eulenburg found it in 28 of his 64 cases, 21 showing double, 7 single myosis. Of my 20 cases, 7 had distinct myosis, and of these 4 had one pupil smaller than the other. The two pupils are seldom exactly of the same size. The degree of contraction varies greatly in different cases; it also varies from time to time in the same case, becoming, however, as a rule more marked as the disease advances, although not necessarily in a regular ratio. It may become very intense, and then again diminish. Thus I have seen it increase during gastric crises; and Charcot,<sup>1</sup> in his admirable lecture on the subject, states that during the attacks of lightning-pain the pupil dilates. He has also remarked that in an early stage of ataxia, it is commonly observed in inequality of the pupils, and on the side on which contraction is more marked there are sometimes evidences of vasomotor paralysis, in the reddened cheek, the congested conjunctiva, and the local elevation of temperature.

In the patient whom I now present to you, you observe extreme myosis. You notice that his ataxia is now far advanced. He often suffers from lightning-pains, and has almost constantly a broad girdle pain around the lower part of his throat. His sensibility is much diminished, patellar tendon-reflex is lost, and he has the characteristic ataxic gait, and sways about and staggers when he attempts to stand with his eyes closed. You observe that his pupils are like pin-holes, and they have been so for years. The myosis was in his case an early symptom; indeed, it was on account of dimness of

<sup>1</sup> 'Leçons sur les Maladies du Système Nerveux, Anomalies de l'Ataxie Locomotrice,' p. 56.

sight that he first sought medical advice. You will understand how such a condition necessarily makes the vision dim, even if there be no change in the optic nerve. If you will hold in front of your eyes a metal plate, or a card, with a very small aperture in it, such as might correspond in size to the pupil of our patient, you will find that although objects may be well enough defined, they appear dim from defective illumination.

Observe also in the left eye of the patient the result of the instillation of atropine. Instead of the complete dilatation produced in the normal eye, we have induced in our patient something less than medium dilatation. This modified effect has long been known as one of the characteristics of spinal myosis.

Myosis is due to disease of the cilio-spinal region of the cord. From that region there proceed nerve-fibres which join the sympathetic, and ultimately supply the radiating muscular fibres of the iris. When from affection of these cilio-spinal nerve filaments the radiating fibres of the iris are paralysed, there is nothing to oppose the action of the circular fibres, which by their contraction produce myosis. The pupil-symptoms of certain cases of thoracic aneurism are explained by pressure on the cilio-spinal fibres.

γ. Associated with the myosis, but sometimes occurring independently of it, is another curious feature, which was first described in 1869 by Dr. Argyll Robertson,<sup>1</sup> and which for convenience we are accustomed at the bedside to call "the Argyll-Robertson symptom." It consists in the absence of any contraction of pupil on exposure of the eye to light, while movement with accommodation is normally retained. This circumstance had not attracted attention until it was described by Dr. Argyll Robertson; and even now it is less known than its importance deserves. It may be well that I quote to you the description as it was originally given.

"That although the retina be quite sensitive, and the pupil contract during the act of accommodation for near objects, yet an alteration in the amount of light admitted to the eye does not influence the size of the pupil."

<sup>1</sup> 'Edin. Med. Journ.' Dec. 1869.

It is much more difficult to find a satisfactory explanation of this fact than of the myosis. Dr. Argyll Robertson suggested an hypothesis that the contraction of the pupil to light is not entirely due, as has been generally supposed, to contraction of the circular fibres of the iris produced through the third nerve but in part, at least, to temporary arrest of action in the radiating fibres of the iris, effected through the cilio-spinal filament, in the sympathetic. This hypothesis demanded that a function be assigned to these last-named fibres which is not known to exist in any other nerve. The considerations which induced him to hazard the hypothesis were the circumstance that, in the cases of spinal myosis which he recorded, the patients retained good vision, indicating a sensitive condition of the retina, normal conducting power in the optic nerve, and healthy state of the brain centre connected with visual impressions; further, that in them the contraction of the pupil associated with the accommodation indicated a healthy state of the third nerve and circular fibres of the iris, and yet, with an apparently complete chain of connection between the sensitive retina on the one hand, and the contractile fibres of the iris on the others light did not affect the pupil. The one pathological condition which was evidently present was paralysis of the cilio-spinal filaments in the cord, to which, therefore, he ascribed a share in the mechanism of pupil contraction under light. According to this view, one lesion seems to explain both the myosis and the Argyll-Robertson symptom.

But it was at once apparent that if this hypothesis were correct, myosis and the Argyll-Robertson phenomenon must always coexist, and I looked anxiously for evidence as to whether this was so or not. The first case in which I found them dissociated was one recorded by Wernicke, in Virchow's *Archiv* for 1872, vol. lvi. The patient was the subject of chronic alcoholism, and his pupils were of normal size, the right larger than the left; the former measuring 2 lines in diameter, the latter  $1\frac{1}{3}$ . The right reacted to light, coming down to  $1\frac{1}{4}$  lines; the left did not react to light, but both acted with accommodation. Now it is true that in this case the pupil which manifested the phenomenon was smaller than the other, but it was not by any means very small. Since that time I

have met with at least one well-marked instance in my own practice in which the pupils were of normal size and were equal, but the phenomenon was distinctly present. It thus appears that this suggestion does not afford the desired explanation.

Wernicke and Hempel adopt the view that the nervous connection between the optic and oculomotor nerves is interrupted,<sup>1</sup> and consequently the reflex irritation of the oculomotor can no longer occur, but the centre of the third is not destroyed. It appears, on the whole, most probable that the movements of the iris on exposure to light, and those for accommodation, are controlled by different nerve centres, and that in this disease the one centre and not the other is affected. Further anatomical and physiological investigations of course may be expected to clear up this question; and I have some hope that certain preparations in my possession may contribute to this end.

As to the frequency of the occurrence of this phenomenon, I know of no statistical statement, but of my 20 cases it was present in 8. In 7 of these it was associated with myosis; in 1 not, the pupils being in that case of normal size. It is not present in every case of myosis. It seems not improbable that its absence in such cases may depend upon its being a later symptom in the order of development.

### III. *Amblyopia and Amaurosis.*

Dimness of sight is also a very common symptom, but it rarely goes on to complete blindness. Besides the degree of blindness arising from the cutting off of rays by the contracting iris already referred to, we find in some cases another form of dimness, temporary and apparently functional, and corresponding to the temporary paralyses so often seen in the earlier stages of the disease. I found, for example, that a man who was under my care in 1872 had suffered from an attack of dimness of vision eight years before, and another eighteen months before I saw him, both attacks being temporary and unexplained. In 4 out of my 20 cases similar facts are noted.

<sup>1</sup> Graef's 'Archiv,' 1876, vol. xxi., Part I., p. 22.

It seems safe to conclude that such temporary seizures depend, as do the temporary muscular paralyses, on some undetermined condition.

But far more important is the permanent and slowly advancing amblyopia. Eulenburg found it in 31 of his 64 cases, and it was present in 14 of my 20 cases. In 7 of the 14 it appeared to be one of the earliest symptoms; in 5 of the cases there was distinct atrophy of the optic disc.

This condition is well illustrated in the patient before you. The sight of his right eye has been gradually diminishing, and is now quite lost, so that he cannot distinguish light from darkness. The sight of the left eye is also impaired. The ophthalmoscope reveals in both of the discs marked atrophic change, most advanced, of course, in the right. The features of this condition are admirably described by Charcot and sketched by Erb. I quote the description given by the latter (*Ziemssen's Cyclopedia*, Eng. Trans. vol. xiii. p. 579).

"But the atrophy of the optic nerve, which is unfortunately very frequent in tabes, is of much more importance than the disturbances of the visual apparatus thus far considered, and is much harder for the patient to endure. It belongs to the saddest complications of this already melancholy disease; the helplessness caused by the ataxy is aggravated beyond all measure by the blindness added thereto, and the sources from which the patients can obtain comfort and oblivion amid their suffering are materially diminished.

"The trouble begins with slowly or rapidly advancing diminution in the sharpness of vision, which soon increases to amblyopia, and finally to amaurosis. The field of vision, which is at first slightly veiled and cloudy, becomes increasingly narrowed, generally from without inward, sometimes from without and above, sometimes more from below. Examination shows that this narrowing of the field of vision does not take place in a uniform manner, but with entering angles. Finally, the blind area involves all but a spot towards the inside, which allows the patient but a limited and insufficient amount of vision. . . ."

"Ophthalmoscopic examination reveals the signs of so-called white atrophy of the optic nerve. At first there is a slight



grayish discoloration of the papilla, which gradually grows paler, and finally appears quite white and sharply outlined. The arteries at the same time show a progressive narrowing, but otherwise the retina remains quite unaltered. These changes are easily to be distinguished from the ophthalmoscopic picture of neuritis optica and congested papilla, and, according to French authors, they are so characteristic that the existence on the approach of tabes can be recognised from them alone.

“Atrophy of the optic nerve in tabes is always—with quite rare exceptions—a decidedly progressive malady, and leads, without interruption, to complete amaurosis. In different cases, it is true, this occurs with very variable rapidity; sometimes it requires only weeks, sometimes months or even years, for its accomplishment. Occasionally the malady comes to a standstill, even after it has lasted for a comparatively long time. Sometimes the trouble is limited for a considerable time to one eye, but it is far more common for both to be attacked and to become blind simultaneously, or within a short time. Modifications between these two extremes are of course manifold.”

Such dimness of sight and white atrophy of the nerve is by no means confined to locomotor ataxia, for it sometimes occurs without any central lesion, sometimes with cerebral disease, and sometimes with other spinal affections.

In illustration of this, I show you a patient suffering from antero-lateral sclerosis, who has for upwards of a year had well-marked amblyopia, with white atrophy of the optic nerve. In all these cases the anatomical change is essentially the same with that which exists in the spinal cord.

#### IV. *Colour-Blindness.*

This is another symptom, fully recognised as occurring in locomotor ataxia. You have seen recently, in Ward I., an illustration of this condition, and I shall presently show you a well-marked example. It is not so common as the other eye-symptoms. Of my 20 cases it was noted only in 3. One of them is the patient now before you. His colour-blindness has

existed for a long time, and it appears to vary from time to time. Thus, in February 1877 he could recognise green, white, pink, and black; but scarlet he called yellow. A year later he called green, dark-brown; red, black; yellow, he called light, but not white; and in February 1879 he could discern blue, yellow, and red. I now show him a series of sheets of coloured paper; the blue, he says, is white; the red is like blot-sheet, the green is drab, the pink is white, the yellow is yellow, the orange-brown is yellow, and the white is recognised as white. He is thus considerably worse than he was four months ago.

It is stated by Erb that colour-blindness can usually be demonstrated before any limitation in the field of vision, and he states that generally the perception of green is lost first, then that of red, finally that of yellow and blue.

Charcot states that the achromatopsia is characterised by loss of perception of the secondary colours, and by loss of perception of red and green; perception of yellow and blue persisting in a high degree, and for a long time. The facts I have shown you in our patient do not strictly correspond with these statements.

It is clear that colour-blindness does not necessarily accompany the amblyopia of locomotor ataxia, and we have not at present sufficient evidence to determine the question, whether it is more frequently associated with this form of amblyopia than with others. I have also no knowledge of any case in which colour-blindness alone was present, and am therefore inclined to attribute it to some peculiar distribution of the atrophic condition in the optic nerve.

I have not deemed it necessary to draw your attention specially to other eye-symptoms which have been described, because they are certainly of very rare occurrence, and, so far as I can judge, of but little importance.

## REMARKS ON BULBAR PARALYSIS, WITH SPECIAL REFERENCE TO ARTIFICIAL FEEDING.

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THE consideration of this subject is to my mind of such great importance, that I have no hesitation in bringing it before the readers of 'BRAIN,' because I do not find that it is insisted upon in those papers and articles that have come under my notice relative to the treatment of this disease. For instance, in Erb's article on "Progressive Bulbar Paralysis" in Ziemssen's 'Encyclopedia of the Practice of Medicine,' we find the following :

"When deglutition becomes impossible, we must feed the patient through a flexible tube ; but with many patients this is rendered impossible by violent fits of coughing, vomiting and suffocation. In this case nothing is left but the administration of nutritive enemata, such as solution of pancreas, milk and eggs, bouillon, wine, &c."

For many years past I have, in speaking or writing on the treatment of this affection, been particularly careful to draw attention to the necessity that arises for artificial feeding *before deglutition becomes impossible*, and as soon as it is in any way interfered with.

I do not wish it to be inferred from this that no nourishment should be allowed to pass into the stomach except through the tube, but I would have the bulk of nutritive material introduced in this way. The call for nourishment and the rapacity of the individual is astonishing ; and as the difficulty in

swallowing increases, so does the desire for food, and in direct ratio.

It must be understood that the nature of this communication is practical even to commonplace, so that its real meaning shall be at once grasped even by those who do not care to study psycho-physiological problems.

The connection between the medulla oblongata and the bulb with the cord below and the central nervous system above, is recognised even by advanced scientific investigators of the present day to be of the most complicated nature; and when we consider the immediate connection of the medulla oblongata with the entangled web of fibres in the pons, we are at no loss for evidence to show how difficult it not unfrequently is to make a correct diagnosis in reference to pontine and bulbar lesions. We can thus see how readily many observers are and have been led into error, and that many palsies, including those of the muscles of deglutition, which have been called bulbar paralyses, are really not so, but take their origin from some disease either below or above the medulla oblongata. I know of no published record at the present time where this differential diagnosis has been carried out with anything like completeness and precision, although many valuable articles written upon this subject are to be found.

In all probability this is due to our want of knowledge of the connection between the histological elements and the course of the nerve fibres in the medulla oblongata. Erb says:

“Notwithstanding all the investigations that have been made, and the incalculable trouble expended upon this subject, we are scarcely acquainted with the barest outlines. We know just as little about the fate of most of the fasciculi which enter the medulla from below and from above as we do of the significance of numerous striking structures in the medulla. Their connection with one another and with the gray nuclei, the mode of interruption and where it occurs, their transposition and their termination, all remain a mystery.”

But the study of the bare pathology of medullary disease does not give us so much trouble as tracing the histological connection of its bundles of fibres, and provided we find

clinical signs which are indicative of lesions of those nerve centres which exist in the floor of the fourth ventricle in immediate connection with the bulb, we are satisfied, as far as our clinical work is concerned.

But perhaps of the two conditions we are as physicians more immediately interested in the vascular supply of the medulla oblongata as productive of pathological changes.

M. Duret<sup>1</sup> has shown that the medulla receives its blood supply from the vertebral and basilar arteries, the median branchlets which pierce into the raphe between the pyramids, and are given off by the art. spinalis for the bulbus and by the basilaris for the pons. But besides these, small vessels pierce the medulla along with the nerve roots, and run to the corresponding nuclei, where they take part in the formation of the capillaries. The olivary bodies and pyramids are supplied by small branches from the vertebral and anterior spinal arteries. The restiform bodies receive vessels from the art. cerebelli infer. post., whilst the post. spinal arteries send branches to the pedunc. cerebelli and floor of the fourth ventricle. I have always maintained—and my views are confirmed by experience—that whenever we have direct disease of the bulbar nerves and nervous centres, it is invariably due either to tumour, to change in the coats of the vessels, or to the production of thrombi within the vessels themselves.

I have no hesitation in saying that bulbar paralysis, pure and simple, is one of the rarest forms of disease of the nervous system with which neurologists are acquainted, and that it is more frequently than otherwise associated with progressive disease of the optic thalami, crura and pons from above, or with ascending disease of the spinal cord.

The troubles of speech are not necessarily associated with bulbar disease, but, as M. Voisin has pointed out with good show of reason, where the speech is of a jabbering, stammering kind, it is usually the result of disease of the medulla oblongata.

Many cases which have been designated labio-glossolaryngeal and pharyngeal paralysis, and stated to be due to

<sup>1</sup> Duret, sur la distribution des artères nourricières du bulbe rachidien, *Arch. de Physiol. et Pathol.*, 1873, p. 97.

disease of the bulbo-rachidian nerves, have not in reality been so. Notably some of those reported by M. Trousseau and Duchenne, and by others as retrogressive disease of the bulb. They have been, in fact, instances with which we are now quite familiar, of primary lesions existing in a given motor convolitional area, or in the basic cerebral ganglia extending progressively towards the bulb, and disturbing and eventually annihilating its function. Yet, for the sake of demonstration, such cases as these come especially under our observation when we have to consider their treatment by artificial feeding. When we note with care the attendant symptoms and signs of bulbar paralysis, such as the affections of speech, mastication, deglutition, expression, respiration, circulation, and the continuous flow of saliva from the mouth, our diagnosis is practically simple. It is foreign to the object of this short paper to differentiate in any way between these signs, but we must glance at the disorders of nutrition, not only with the parts immediately in relation with the bulbar nervous supply, but of the body generally. Atrophy of the muscles supplied by the bulbar nerves is of regular and constant occurrence in this disease; and in almost every case, with some few exceptions, the atrophy is regular, progressive, and uniform; but perhaps it is more marked in the tongue, which looks limp, becomes furrowed, small, flattened, shrunk, and is in a constant state of vibratory motion due to fibrillary contractions. The patient's want of voluntary power over this member is extremely pathognomonic, and the first indication evidenced may or may not be due to defective articulation. There can be little doubt that it is usually so. Yet, as pathologists, we are perfectly aware that considerable disease can exist in the large ganglion cells of the motor nerve nuclei in the floor of the fourth ventricle, prior to any observable changes going on in the muscles supplied by these nerves. Not that the muscles themselves are escaping the inevitable and ultimate changes which must follow, but rather that these changes are not in the first instance, or even for some time, so impaired as to have their normal function objectively interfered with.

And here we come to a part of the subject to which I have always ventured to draw particular attention. In making an

early and correct diagnosis, if we hope that our treatment shall be attended with success, it is necessary to watch carefully the signs of fatigue of the muscles of the lips, tongue, and pharynx: in the first place, from a normal standard in relation to their individual functions; secondly, from a normal standard in relation to their correlative and co-ordinating functions; thirdly, in reference to these in combination with enforced or extraordinary volitional, automatic, and compound co-ordinating functions. By this means we shall be able to ascertain the almost immediate changes, or general nutritive disturbances which take place from those changes, which are undoubtedly of primary origin in the motor nerve nuclei—at all events, in the form of paralysis now under consideration.

So thoroughly conflicting are the statements as to faradic and galvanic irritability of muscle and nerve in bulbar paralysis, that one feels inclined to discard them altogether as sources of error and fallacy; but my colleague, M. de Watteville, certainly seems to have studied the question with considerable care, and to have elucidated some results which are capable of confirmation, if not of practical application in diagnosis. He says ('Introduction to Medical Electricity,' p. 99), "That electro-diagnosis, especially in the earlier stages of bulbar paralysis, points to its connection with other diseases of the grey matter, such as progressive muscular atrophy, and amyotrophic lateral sclerosis. Careful investigation shows in these diseases a diminished electro-muscular reaction to both faradic and galvanic current, with a tendency to a preponderance of the positive over the negative pole. Electro-nervous irritability is not necessarily affected; the disease then differs from poliomyelitis anterior by not giving the full reaction of degeneration, but only the incomplete form of it so well studied by Prof. Erb, of Heidelberg."

Erb writes, Ziemssen's 'Encyclopedia of Medicine,' vol. 13: "Encouraged by my observations on progressive muscular atrophy, and led on by the conviction that the form of degenerative atrophy with which we have to deal in bulbar paralysis would be physiologically expressed by the 'reaction of degeneration,' I expected certainly to find this reaction in the disease, and my expectation was fully gratified by the

first pronounced case which came under my observation, and which I examined with great care. I found in fact the most marked reaction of degeneration in irritation of the muscles both on the chin and in the lips, and even in the tongue. At the same time, however, the electric irritability of their nerves was normal, or but slightly diminished, so that we have exactly the same form of degenerative reaction which I have described as characteristic of the so-called 'middle form' of certain peripheral paralyses, and I have no doubt we shall find the same thing if we look carefully for it in all cases where atrophy of the muscles has reached a certain period."

From personal experience, I have relied little upon either electrical or galvanic phenomena, for it is quite clear that muscles in their natural state vary considerably in their contractile power, either through the agency of the faradic or continuous currents. I would, in making diagnosis, depend chiefly upon the clinical evidence which has been brought to light by enforced fatigue, or strained co-ordinating effort. The act of mastication, though purely voluntary in a healthy state of the centres of the bulbar nerves, becomes less so in proportion as these centres are directly or indirectly involved, and the important part played by the hypoglossal nerve through the muscles which regulate the movements of the tongue becomes so strikingly manifest during even the incipient stage of bulbar disease, that we are compelled here to advert to it, and to the process of deglutition. Complex and voluntary as these movements are, they differ little from other complex movements, in being to a great extent automatic or reflex, and volitional power exercised over them is rather the exception than the rule. Something gets between the teeth, or between the teeth and the cheek, which cannot be easily removed, and the branches of the fifth nerve convey the special muscular sense and contact sensation to the brain, which send forth motor volitional impulses from this centre, to effect by an increased volitional act what the mere reflex automatic act is unable to effect. Now in the act of deglutition we readily admit that we have, as is well known to the student of physiology, processes which are far more intricate and delicate than the mere mechanism of mastication. Guided



by our text-books rather for mathematical exposition than from pure physiological reasoning, we find that the first act of deglutition is said to be voluntary; the raising of the soft palate and the approximation of the post pillars of the fauces are intimately associated with the tongue in passing the bolus of food into the upper part of the bag of the pharynx; but after the food has passed the anterior pillars of the fauces, then the act of deglutition is essentially reflex, and the will is said to have no power over the constrictors either to originate or modify their action. If the bulbar centres are diseased, and if we admit that the reflex centre exists in the medulla oblongata, it must be evident that the act of deglutition becomes not only voluntarily but automatically and reflexly most seriously impaired; and it is not, let it be understood, the first act of deglutition which gives the patient most serious and agonizing distress; but it is in the second and third stages through hyper-reflex and abnormal excitations which are so distressing. And here I am forced to support a doctrine which at times must occur to the practical physician, that normal physiological acts have not unfrequently rather an indirect than a direct bearing upon anomalous diseased states. In some cases of bulbar paralysis we know that reflex actions are strikingly diminished. We can touch, irritate, and apply a strong continuous current to the pharynx, soft palate, and even the larynx, without exciting them. Yet the patients have little difficulty in localising the sense of touch; but such a want of reflex sensibility does not usually exist in that early stage of the disease where we consider artificial feeding to be so supremely essential. If in bulbar paralysis we had to combat the patient's unwillingness and determination not to take food, as we so frequently have to do with insane patients, my paper would fall short of its object; but what I hold is this, that in the form of paralysis which we have under our notice, two things are absolutely essential to its rational treatment, namely, a highly nutritious and assimilable diet to be given at frequent intervals, and as far as possible to keep the muscles of expression, articulation, and deglutition, in a state of psychological no less than physiological rest. This must be at once apparent to the physician who has carefully

noted the onset and progress of these cases. The suffocative cry of despair uttered by the unfortunate creatures when they feel themselves powerless either to eject or swallow the food lying passively on the isthmus of the fauces, or between the gums and the cheeks, tells its own tale. In one case which was under my care, a young fellow, exhibited at the Medical Society of London, improved considerably under treatment. His appetite was simply voracious: he was fed regularly through the nose by means of a piece of india-rubber tubing, which he usually passed himself well into the œsophagus; but, independently of this, he stole food whenever he could, and the only way he got it into the pharynx was to cram his mouth to its utmost limit, and then push the food with his finger down the throat.

Upon his visit to the Medical Society<sup>1</sup> the different processes of nasal feeding were put to the test. Dr. Tuke's highly finished and somewhat catheter-like instrument could not be introduced, but an ordinary piece of india-rubber tubing of less than  $\frac{1}{2}$ -inch diameter passed without any trouble.

A few remarks about artificial feeding in general will conclude my paper, with a word or two concerning nasal feeding.

Feeding the insane through the nostrils was first prominently brought before the profession, and its advantages ably maintained by Dr. Moxey, in the columns of the 'Lancet,' 1869-73, although Dr. Clouston amongst others wrote rather biassedly in favour of the stomach-pump and gag. I think there can be little doubt that the no less scientific, though perhaps unnatural, mode of procedure, of making the passages of the nose a means of direct communication with the involuntary muscles of the œsophagus is now generally admitted and usually adopted. For my part and in my experience, the latter has exceptional advantages of which I have always availed myself, no less in cases where forcible feeding has been required than in those to which this paper especially refers. To prevent reflex irritability, the tube should pass beyond the muscles of the pharynx, then the food enters the stomach in a continuous stream, no matter what efforts are

<sup>1</sup> 'Med. Soc. Proceedings,' vol. i.

made on the part of the patient to prevent it. I cannot but apprehend considerable inconvenience may occasionally arise from merely pouring the fluid through the nostril, unaccompanied by tubing. Concerning the difficulty which sometimes occurs in passing a tube through the nostrils into the oesophagus, I am quite aware that it depends no less upon the skill of the operator than upon the nature and size of the tubing used. The introduction of a bougie, or catheter, through the male urethra, is by comparison a teaching example. We have all found by practice that a No. 10 elastic catheter will glide into the bladder with perfect ease and total absence of pain to the patient, when a No. 4, if passed at all, is accompanied by excruciating pain and discomfort. It is not less so in the passage of the naso-oesophageal tube, and I have more than once been compelled to desist from attempting to pass a highly-wrought beautifully tapering elastic oesophageal tube, when a piece of common india-rubber tubing has been subsequently passed with facility. In forcible feeding, we have on the introduction of the tube not unfrequently to overcome volitional spasm of the pharyngeal muscles, by preventing the access of air to the patient's lungs, when in a few seconds the automatic compulsory respiratory effort soon necessitates a deep inspiration, and the difficulty is at once removed by the immediate passage of the tube. In spasmodic strictures of the urethra, prolonged gentle firm pressure causes the primary spasm to yield, and the obstacle is not only overcome but the temporary muscular inertia produced by overstrain renders the passage of the catheter doubly easy. We find at times, when the operation of nasal feeding is conducted imperfectly and hurriedly, that reflex irritation gives rise to a sense of choking and perhaps sputtering of fluid from the mouth and nose, but this is simply due to the absence of careful manipulation. For some years past my oesophageal tube and funnel for nasal feeding has consisted of an ordinary india-rubber inflation pessary (a hole being cut in the bulb which forms the funnel to admit of the pouring in of the fluid). The patients usually introduce the tubes themselves, and the nurse pours in the fluid nourishment. A man, with dysphagia and an incomplete bulbar paralysis, was artificially fed on this system,

and some few short notes of his case may be of interest. He was under my care for a month, and increased eight pounds in weight, and improved equally in other respects. W. C., aged 63, was admitted into the Central London Sick Asylum at Highgate, on March 22, 1877. It was not an easy matter to get a clear history from him, as his want of intelligence and reasoning powers were more or less at fault. Until five years ago his health was excellent. It appears that in the evening after his day's work, when he was sitting by the fire smoking his pipe, he became giddy and fell from the chair, but without losing consciousness. From this time he never recovered his usual health. There has been more or less weakness in the limbs, but particularly those of the *left* side. His speech and power of swallowing were unaffected by this seizure. In the month of October, four years ago, he had a similar seizure; but it was not until Feb. 1, 1877, that he quite suddenly lost both the power to swallow easily and speaking distinctly. He describes the last attack in this way. He was going to drink some beer, and found that he was unable to swallow one drop of it. On the following morning he tried to work but could not, because his arms felt so weak; he could not swallow, neither could he speak so as to be understood. When I first saw him he was extremely emotional; there was an equal falling of the under lip, and a drawing downwards of the angles of the mouth, with an overflow of saliva, which he was constantly rubbing away with the back of his hand. I was led then to conclude that he was suffering from an affection of the bulb, either direct or indirect. This was soon proved, as we saw by examining the cranial nerves in the following order.

Nerves :—

1st. Sense of smell fairly good; equal on both sides, but not acute.

2nd. Sight has been failing since the first attack. Says that he has often fancied two sixpences were three, and that halfpence were pence. The ophthalmoscope gave evidence of advancing atrophy of the optic discs, and partial obliteration of vessels.

3rd. Normal.

4th. Slight palsy of.

5th. Pterygoids involved. Can depress the lower jaw. No loss of sensation of mucous surface of either cheeks or gums.

7th. Can whistle, but not spit. No flapping of buccinators when he tries to blow, but the column of air sent out is very feeble. When he laughs, the angles of the mouth are well drawn up. Hearing normal.

8th. Says he is a little short of wind. Marked dysphagia. No absence of sensation.

9th. The glossoplegia is incomplete. He can just protrude the tongue over the lower teeth, and raise the tip to the palate. The mucous membrane covering the tongue has not undergone the usual atrophic change one meets with in true bulbar disease. When he gets food into the mouth, he is unable to use the tongue to remove it; but after it has passed through the isthmus of the fauces, it is swallowed without much difficulty.

It must be particularly noted that there is marked absence of voluntary co-ordinating power between the lips and the tongue, and when a voluntary act of the one is attempted, an involuntary act of the other is frequently the result. In reference to speech, there is greater defect sometimes than others. It is usually of the mumbling hesitating character. All the vowels and labials are well pronounced, but the letter C is pronounced "Pthsee," and G, "Dgee." The letter R cannot be articulated. The vocal cords act well, but he cannot talk in a loud voice, or holloa. There is marked inability of the muscles to raise the larynx. The muscular power of both arms is weak, but co-ordinate, and in like manner the legs are affected. He can raise 35lb. with each hand, and 14lb. with each leg when reclining. There is and always has been perfect control over the sphincters. The urine is of low gravity (1010), and free from albumen.

Now it is clear upon casting one's eye over these notes that the signs of paralysis of the muscles supplied by motor nerve fibres, originating in the floor of the fourth ventricle, were in excess of paralysis of those muscles supplied by other motor cerebral centres. Yet we find no absence of sensation which is so usually an accompaniment of bulbar paralysis.

The faradic test was not applied. The precise seats or area of pathological change in this case were undoubtedly a little obscure, and I anticipated they were general as well as circumscribed; but there could be no doubt concerning the very marked improvement which resulted from artificial feeding. I maintain it would have been impossible to have produced so satisfactory a result in so short a time by any other means, and I have brought it forward as one of those cases which proves practically how essentially necessary it is to aid nutrition to its utmost extent artificially, rather than allow our patients to die from inanition and never-ceasing mental disquietude, if not absolute dementia.

## ON AFFECTIONS OF SPEECH FROM DISEASE OF THE BRAIN.

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IN the first\* instalment of this article ('BRAIN,' Vol. I. Part III., p. 304) it was pointed out that there are necessarily numerous degrees and kinds of affection of language, since "different amounts of nervous arrangements in different positions are destroyed with different rapidity in different persons." Moreover cases are vastly different in their different stages; a patient may be quite speechless for a few days, and afterwards improve so as to have at length only slight defect of speech; and of course there are numerous cases of complete recovery. It is necessary to make some division of cases; we roughly made three groups (see p. 314, Vol. I.). We were careful to declare that this division was an arbitrary one, that it was not a scien-

\* I should like to remark that one very general conclusion to which the several facts so far stated, and facts afterwards to be stated, point, was in principle long ago formulated by M. Baillarger. So far back as 1866, 'Med. Times and Gazette,' June 23, I made the following quotations from his writings, which I now reproduce:—

"L'analyse des phénomènes conduit à reconnaître, dans certains cas de ce genre, que l'incitation verbale involontaire persiste, mais que l'incitation volontaire est abolie. Quant à la perversion de la faculté du langage caractérisée par la prononciation de mots incohérents, la lésion consiste encore dans la substitution de la parole automatique à l'incitation verbale volontaire."

I ought to have reproduced this quotation in the first instalment of this article, as evidently I am following pretty closely the principle this distinguished Frenchman has laid down. For the satisfaction of curious persons, I may say that I give it now spontaneously, no one having drawn my attention to the omission. I fear M. Baillarger's acute remarks have attracted little attention, and I say with regret that I had forgotten them. I do not remember from what book I took the quotation.

tific distinction. The divisions usually made are arbitrary too, although the nomenclature being in highly technical psychological and clinical terms, they may appear to the unwary as being real, almost natural, distinctions. As was then insisted on, we must in an empirical inquiry take type-cases; we follow the plan which is tacitly, if not avowedly, adopted in every work on the "Practice of Medicine" with regard to all diseases.

We took for first consideration the simplest group—cases of Loss of Speech (No. 2), p. 314; Cases of Defect of Speech (No. 1), and that deeper involvement of language in which emotional manifestations (No. 3) scarcely remain, are more difficult, and will be considered later. We take the simple case for investigation first, just as, were we writing on hemiplegia, we should take first the simplest case of that paralysis, not the more difficult case, in which there are deep loss of consciousness and lateral deviation of the eyes and head, as well as paralysis of the face, tongue, arm and leg. A patient who loses speech may regain it; it is convenient to consider cases of permanent speechlessness.

On pp. 316 *et seq.* we made some brief general statements as to the speechless patient's condition; we especially insisted on the necessity of recognising a positive as well as a negative element. I feel convinced that unless in all degrees of affection of Language we recognise that the symptomatic condition is duplex, we shall not trace relations betwixt them, and shall be misled into supposing that cases are different in kind when there are only differences of degree. We have remarked on this, p. 316. Further, we shall not be able to trace analogies betwixt these examples of Dissolution beginning in the lower cerebral centres, and cases of Dissolution beginning in the highest centres, that is to say, cases of Insanity where the condition is manifestly duplex. The most important thing showing the duality of the speechless man's condition is given very generally by saying that Speechlessness does not imply Wordlessness. We stated that there is not evidence that the process of perception is damaged in itself; we say "in itself," admitting that perception may suffer from lack of co-operation of speech-use of words.



We have now to consider more particularly the condition of the patient we call speechless. In most cases there remains some utterance. But to utter words is not necessarily to speak. To speak is to propositionise. We admit, however, that in some cases which we call loss of speech there is a recurring utterance, viz. "yes" or "no," which is of propositional value; and that occasionally there occur utterances which are of propositional value, and are made up of several words. But in these cases the recurring utterances which have propositional value are so very general in their application, and the occasional utterances which have propositional value are so rare and usually also of so very general application, that it is almost pedantic to say the patients are not speechless. Nevertheless there are exceptions to our statement that there is *loss* of speech in the type-case (No. 2), and full consideration will be given to them.

We divide the utterances into two classes, Recurring and Occasional.

(1.) *Recurring Utterances.*

Soon after the attack, there may be no sort of utterance. But almost always one comes in a few days or weeks. I used to call them "Stock Utterances." They are always utterable; and they alone remain, with the exception of the rare occasional utterances. We make four divisions of Recurring Utterances.

(1) It is sometimes jargon. In one case it was "Yabby," in another, "Watty." Sometimes there is a succession of different jargon; in one case, "Me, me committimy, pittymy, lor, deah." The patient utters his jargon any time. If he "says" anything, it is always "Yabby," or whatever his jargon may be; in reality he *says* nothing with these utterances; they have no propositional value whatever.

(2) Sometimes the utterance is, what to a healthy person is, a word, as "man," "one," "awful," &c. Such a word is, for use, no better than jargon in the mouth of the speechless patient; it is not a word to him; "man," as a recurring utterance, is not a symbol for a human being. The so-called word comes out, just as "yabby" does, and means no more,

means nothing. A single word might have, in a healthy person, propositional value. For example, were a person asked how many oranges he would buy, the reply "one" would be a proposition. (See p. 312.) But the speechless man's recurring "one" comes out whenever anything comes out, and applies to nothing at all.

Here, having given some examples, I may make one general remark about all kinds of Recurring Utterances. The rule is that the particular recurring utterance each person has at first never changes. Sometimes, however, but exceedingly rarely, it changes. A patient of mine for some months, when under my care in the London Hospital, could only utter the word "Dick." Later it changed to "Jimmy," with the variations of "Jim" and "Jigger." Another general remark is that although these rags and tatters of what was once the patient's speech are of no use as speech, they serve as parts of emotional manifestations; it is rather, we should say, the tones in which they are uttered; it would be most correct to say the patient "sings" his recurring utterance—variations of tone with healthy speech being rudimentary singing (Spencer).

In this service of these, as also in that of the other recurring utterances, we have evidence that Emotional Language is not affected.<sup>1</sup>

The way in which speechless recurring utterances serve patients is exemplified in some cases noted in the Hospital Reports of the *Lancet*, February 17, 1866, and July 20, 1867. Several other things of importance for other departments of our subject are given in the following extracts.

"The patient we saw could only utter the word 'Dick,' and this word he uttered whenever we asked him a question. We were told that when the man was vexed by the other patients in his ward he would swear. He generally used the common explosive sound, so much in favour with English swearers. He could not, however, say the word when required to do so, even whilst it was well kept before his mind by frequent repetition. He seemed to make efforts to say it, but the word

<sup>1</sup> I would not affirm that the finest emotional manifestations may not be lost in cases of loss of speech; I do not know that they are. It would be very remarkable if they were not.

‘Dick’ always came out instead. The oath was only uttered under the influence of emotion, and could never be repeated at will.”—The *Lancet*, Feb. 17, 1866.

The following is a further note of the same case from the *Mirror of the Lancet*, July 20, 1867.

“When the poor fellow left the London Hospital he was able to utter the word ‘Dick’ only, except that he swore when vexed. He is now in a workhouse, where, thanks to the permission of Dr. E. H. Moore, Dr. Hughlings-Jackson saw him a few weeks ago. Strange to say, the patient’s stock phrase is now ‘Jimmy;’ he never says ‘Dick.’ Although it is two years since the patient left the hospital, as soon as he saw the doctor he raised himself eagerly from his chair, offered his left hand—his right is still paralysed—and cried out very vivaciously, ‘Jimmy, Jimmy,’ &c., evidently pleased to see some one whom he knew. The ward superintendent says the patient sometimes sings; that the word he then uses is ‘jigger.’ He is usually quiet; but when vexed he swears, or rather utters a very nasty word, the last syllable of which rhymes to the last syllable of jigger. He cannot say this word when he tries, but, when trying, says ‘Jim’ instead. ‘Jimmy’ seems to be the word he uses as an ejaculation to show states of feeling, and ‘Jim’ when he is trying to convey information. When asked to show how many children he had, he extended his left five digits twice, and at each extension he uttered jerkily the word ‘Jim.’ At a second visit, the man replied to the same question in the same way; but there are no means of knowing whether his reply is a correct one or not. He does not tell the number of days in the week by this plan. He sang when asked; and although the performance was of the very poorest kind, there was cadence with variation of tone. In one of these efforts he used as a vehicle of sound the word ‘Jim,’ in another, ‘jigger.’ The ward superintendent remarked that the man’s friends had not visited him since Christmas. Here the patient clenched his fist, tightened his lips, face, and neck, holding his breath, and turning red the while, as if making an effort. After a moment or two he sighed deeply and relaxed, shook his head, and looked as if he had given up an attempt to do something. The attendant believed the

patient was 'trying to talk,' and said he often saw him put himself in that way. As Dr. Hughlings-Jackson was leaving the room, the patient left by another door, but in passing through the doorway he stopped, and turned his head as if he had suddenly remembered something, looked towards the doctor, and said pleasantly, 'Jim, Jim.' It was supposed that this meant good-bye."

I may here mention that I did not get to know if the patient had sons of the name of Richard and James (*vide infra*).

The following is from the same *Mirror* of the *Lancet* as the last quotation. It shows a certain *use* of the jargon "ow," during the expression of number, as well as its use as the proposition "yes."

"In another workhouse Dr. Hughlings-Jackson saw, with Dr. Edward Richardson and with his assistant, Mr. Widdas, a woman twenty-five years of age, who is only able to utter the phrase 'Oh! my God!' and the noise 'ow'—probably a corruption of oh! When the doctors went up to her bed and spoke to her, she cried out 'Oh! my God!' When next spoken to, she said 'Oh!' and then put her hand over her mouth. She uttered the phrase several times in the interview; but she 'spoke' with the syllable 'ow,' expressing assent or dissent by the tone she gave to it, and by her manner. She was asked how long it was after her confinement before the loss of speech came on. She held out her five left fingers, and said 'ow,' and then separating one finger from the rest of those of the paralysed right hand, again said 'ow.' The doctors said interrogatively, 'Six?' She nodded, and said 'ow.' They then asked whether weeks, months, or years, really themselves knowing the right time. By variation of tone of 'ow,' by nodding and shaking the head, she expressed assent or dissent when the right or wrong period was named. She laughed heartily when something jocose was said, crying out 'Oh! my God!' When the death of her baby was mentioned, her eyes filled with tears. The nurse says the woman was once in her ward before, and then the words uttered were 'Oh! my goodness will!'"

(3) The Recurring Utterance is sometimes a phrase. In one case "Come on," or sometimes that patient uttered "Come

on to me." In another case, just mentioned, it was, "Oh! my God!" In another case, mentioned to me by Dr. Langdon Down, "Yes, but you know."

In some cases, as in the one first mentioned, the patient may utter "yes" or "no," or both, in addition to his recurring phrase—see (4). He has then two sets of recurring utterances.

These phrases, which have propositional structure, have in the mouths of speechless patients no propositional function. They are not speech, being never used as speech; they are for use only compound jargon; they or their tones are at the best of interjectional value only. The man who uttered "Come on to me," uttered it on every occasion when he made a rejoinder to anything said to him.

(4) A common thing is that the patient retains as his sole utterance "yes" or "no," or both these words. Sometimes there is in addition some utterance of one of the other divisions. This must be carefully borne in mind. We shall consider the utterances "yes" and "no" at length. It is the most important part of the whole inquiry. The consideration of these and of some other fundamentally like phenomena will help us out of the empirical stage of divisions into the scientific one of distinctions.

To speak is, as has been said, to propositionise; many verbal utterances by the healthy are not speech. Now the words "yes" and "no" are propositions; indeed to call them "words" is not to acknowledge their proper rank; "proposition-words" might be a more correct expression. But they are not always propositions—are not always used for assent and dissent; and thus the term "word" is convenient, if not strictly accurate.

It does not matter what the philological history of the words may be; at any rate "yes" and "no" stand for propositions. They are propositions in effect; we can *say* with them. Nor must we limit ourselves to the very syllables "yes" and "no." One of my patients had the utterance "Eh," which was "yes" for him, and possibly was a corruption of his healthy "yes." Were we now dealing with the less special part of intellectual language, pantomime, we should admit nodding the head for assent and shaking the head for dissent to be pantomimic propositions. With the other recurring utterances, (1), (2), and

(3), the patient says nothing; they are a mere series of syllables; the so-called words and phrases (2 and 3) being intellectually dead. Is not there then in the utterance of "yes" and "no" a real exception to the statement that our patient is speechless?

An utterance is or is not a proposition according as it is used.

(a) The speechless patient may utter "yes," or "no," or both, in different tones, merely according as he is thus or thus excited. It is then not a proposition, but an interjection, a mere vehicle for variations of voice, expressive of feeling. (b) He may have this service of the words and be able also to reply with them; the latter is a propositional use of them. (c) He may (in addition to (a) and (b)) be able to say the words when told to say them.

(a) A speechless patient may utter "yes" and "no" without any sort of application. He may utter "yes" when he means "no," and "no" when he means "yes." He may nod when he utters "yes." He may affirm or deny by the less special language of pantomime when he cannot *use* for affirmation and denial the words of affirmation and denial which he can glibly utter. They are not, therefore, propositions to him. Nevertheless, this low degree of the utterances serves him. He utters "Yes," "yes," "yes," or "No," "no," "no," merrily, or he utters them sadly, when respectively glad or sorry. That is to say, although he has not the propositional use of "yes" and "no," there is that emotional service of them which other speechless patients have of their recurring jargon, words or phrase (1, 2, 3). His utterances of them in various tones are revelations to *us* of his varying emotional states. We must be careful not to give *such* utterances of "yes" and "no" the credit of being propositions. From the tones in which they are uttered, *we* may understand or guess how the patient is feeling; and with the conspiring aid of the then circumstances, we or his friends may often *infer* what he is thinking. But so we could by the tones in which the recurring jargon (1) or phrase (2) is uttered. Like smiles, they are, when so used, not signs for emotional states, but they are, or rather the tones of them are, *parts of* this or that emotional manifestation. On the other

hand, if the patient who generally uttered "no" at random, *used* "no" in a particular tone, in order to signify that he dissented, it would be speech, or at any rate of speech-value; and so it would be speech or of speech-value if a speechless man used his jargon with the same intention. The woman who uttered "ow" (p. 208) could express assent or dissent by the different tone she gave to it. The following is a striking case.

I have seen a patient who nearly a year before had become rapidly apoplectic. On recovering from this condition, he uttered only "low," but soon he uttered "no," and when I saw him he had nearly recovered speech. His articulation was defective, but his wife could understand what he said, not merely guess his meaning, and I could nearly always do the same. He could express himself in writing, and could read. For the moment using popular language (*"without prejudice"*), he had the mental power of speech, but had defect in the executive. But he very often uttered the word "no" when he meant "yes:" this is a very rare thing in the midst of so much recovery of speech. In reply to one of my questions, he uttered "No," "no." His wife said he meant "yes:" he nodded. Later in our investigation he uttered "no;" but his medical attendant, alive to his misuse of that word, said, "Do you *mean* 'no'?" The patient showed that he did by re-uttering it in a ceremonious, slow, decided, tone. Thus the patient *uttered*, in a tone to signify dissent, the word which, as usually uttered by him, would not have meant dissent.<sup>1</sup>

Here plainly "no" was not a proposition, but the tone it was uttered in was of propositional value—at least vocal pantomime. It matters not what trick or dodge (tone of voice, cardsharp's smile, &c.), be used to express assent or dissent, or to express any relation betwixt things; if so used, there is a proposition.

<sup>1</sup> Tylor says that in some languages, "especially in South-East Asia, rises and falls of tone to some extent, like those which serve us in conveying emphasis, question and answer, &c., actually give different significations. Thus, in Siamese, há = to seek, hā = pestilence, hà = five. The consequence of this elaborate system of tone-accentuation is the necessity of an accumulation of expletive particles to supply the place of the oratorical or emphatic intonation, which, being thus given over to the dictionary, is lost for the grammar."—'Primitive Culture,' vol. i. p. 153.

Some years ago I had under my care in the London Hospital a man whose sole utterance was "no," and something like "eh," which was "yes" to him. His wife told me he could make the children "behave when they were at the top of the yard" by shouting out "No," "no," "no," in an angry tone. These emotional utterances of his may seem to have some slight propositional flavour; he may have *used* "no" in an angry tone, not merely uttered it during vexation.<sup>1</sup> This, however, is doubtful. The patient could reply "no," but the slight degree of his power of expressing himself may be judged of by his way of getting his children to understand what he wanted. He would make one stand before him; she would guess one thing after another until, by quickness or by lucky accident, she guessed what he wanted, or until he knocked her down with his fist. I shall have to refer to this case several times, and shall therefore call the patient "Dow."

(b) In some cases of loss of speech there is a use of the words "yes" and "no," which is higher than a mere emotional service. The patient can reply with them. Here then is evidence that the so-called speechless man is not absolutely speechless; he propositionises by "yes" and "no." Now we come to a very important matter. In the case of "no," at least the use of that word does not in all patients reach the level of normal speech: or, speaking more correctly, the patient cannot utter that word in all the ways healthy people can. He may be, as aforesaid, able to reply "no" to a question requiring dissent, when he cannot say the word when he is told and when he tries. This has been observed and commented on by Sir Thomas Watson, in the last edition of his 'Practice of Physic.' I found it out when giving a clinical demonstration of "Dow's" case. I told the students that he could utter the word "no;" but, to my chagrin, when I asked him to utter it, there was nothing but an articulatory effort. However, on asking him the preposterous question, "Are you ninety years old?" the word "No" came out at once. Again I asked him to "say no;" his efforts were fruitless, but we readily got the word out

<sup>1</sup> If a speechless man retains the two words, it is reasonable to suppose that the emotions of fear and anger would, to speak figuratively, appropriate the negative one, and that emotions of joy and sympathy would appropriate the affirmative.



of him again by asking another question, which obviously required a reply of dissent. His difficulty was not from nervousness: his wife had found out, before I did, that he could not say "no" when he tried.<sup>1</sup>

I find that I have led Kussmaul to misunderstand me on this matter. This distinguished physician writes: "Jackson and Sir Thos. Watson have even found that aphasic persons, unable to reply 'no' to a question, have nevertheless been led to do so by suggestions designed to make them angry, e.g. 'were they a hundred years old, or a thousand?' or such like." The questions were not designed to make the patients angry, and did not make them angry. The patients I speak of could reply by "no" at any time. A very preposterous question was asked in order that there might be no possible doubt that a negative was required. And I submit that the rejoinder "no" to such a question by the patient "Dow" was a reply, that it was "no" as a proposition; that it was speech, although inferior speech (not incomplete speech); and that it was not "no" as a mere utterance, like an oath coming out in anger. This patient, as I have stated, did use the word emotionally when vexed; but not so in rejoinder to the preposterous questions I asked him.

(c) In many cases of loss of speech, the patient, besides having the emotional service and also the power of reply with the words, can say "yes" or "no" when told (he has the full use of) these words. It may be said that this third degree of utterance of the word is not speech; that it is uttering the

<sup>1</sup> The following is from the 'Hospital Reports' of the 'British Medical Journal', December 2, 1871, and refers to a case of loss of speech: ". . . She was told to say 'No,' and could not. Directly afterwards, Dr. Hughlings-Jackson, observing she had a book on her lap, asked if the patient could read. Hearing this, the patient herself looked up and said, 'No, no, no.' She was again told to say 'no'; she could not. The nurse, having observed this peculiarity in another patient, said, 'Are you a hundred years old?' The response was 'No,' with a smile. Once more the patient was asked to say 'No,' but again she failed. . . ." It must be added that in her ordinary "conversation" she sometimes said "no," when she meant "yes." In some cases there is no difficulty in saying "no." Thus there is in the London Hospital an old woman who can utter only the words "yes" and "no," and "titty," "titty;" and another old woman who has both these words and the utterance "I'm very well," or "Very well." Each of these patients can say "no," when asked. Hence it is admitted that some aphasics have the full use of these words.

word as an articulatory gymnastic "for the sake of uttering it," not *using* it as a proposition. There are weighty reasons, however, for drawing attention to the three degrees of utterance of this word. The inability to say "no," when told, with ability to utter it in reply and also emotionally, is one of the most important facts in the matter of affections of speech. I shall speak on this matter after considering analogous peculiarities.

To resume. In some cases called *loss* of speech there is not absolute loss. The utterances (1) and (2) and (3) are not exceptions; the utterances "yes" and "no," in reply, are exceptions, these words being *used* as propositions.

These exceptions are very significant. The man has lost all speech, except the two most general, most automatic, of all his propositions. They are indeed very significant exceptions to the empirical division into loss of intellectual and conservation of emotional language; for, even regarded superficially, they stand on the border ground. These words are used by healthy people, now one way, now the other; they are sometimes parts of emotional manifestations, and may then be combined with an ordinary interjection, as in "oh! yes," or be duplicated as "no, no;" here the second "no" at any rate is interjectional. They are at other times used with full and definite propositional intent to signify "this is so," or "is not so." The word "yes" may be used at the same time, both for sympathy and agreement, it being occasionally hard to say whether the intellectual or the emotional side is more visible. Similarly some movements are at once pantomimic and gesticulatory.

We hear these words used nearly purely emotionally, very often. A woman suffering from pleurisy and in great distress, replied "no" to a question, used the word propositionally, and then went on uttering the word as a vehicle of tone, "No, no, no," in just the same way as she had been uttering "oh!" before the question; that is, it served her emotionally. A healthy man, told suddenly a piece of startling news, cries "No!" using "no" not actually to deny the truth of the statement; indeed he does not use it as a proposition; it is an ejaculation of surprise, equivalent to the exclamation "Non-

sense!" or "You don't say so!" These so used are interjections, not speech, and take low rank in language, little above that of bodily starts, parts of common emotional language. At the best they are propositions, entirely subordinated to the service of an emotion.

Then propositionally "yes" and "no" give assent or dissent to anything whatever; they are the blank forms of, or stand for, all negative and positive propositions—are, as it were, propositions almost reduced to positive and negative copulas. From their almost universal applicability they are very frequently used; they are the most general, most automatic, and most organised of all propositions. They are then exceptions proving the rule; the patient has lost all speech except these two propositions, which are at the "bottom" of Intellectual Language and at the "top" of Emotional Language. In other words, the retention of these two words is not exceptional to the principle of Dissolution. The reader may, however, urge that the other recurring utterances are exceptional. I hope to show, later on, that they are not. At present, I only say that I believe them to represent what was, or to represent part of what was, the last proposition the patient uttered or was about to utter when taken ill.

## (2) *Occasional Utterances.*

These utterances are rare, except that some patients swear very frequently. We shall make three degrees of these ejaculations:

- (1) Utterances which are not speech;
- (2) Utterances which are inferior speech;
- (3) Utterances which are real speech.

(1) Under excitement, the speechless man may utter "Oh!" or "Ah!" More than this, he may swear, or utter certain nasty words used by vulgar people when excited. (We use the term swearing in the wide sense of what is popularly called bad language; of course religious commination is not considered.) The occasional utterance may be an innocent ejaculation, as, "Oh dear!" or, "Bless my life!" None of these utterances are speech;

they have no intellectual meaning. Moreover, the patient cannot repeat them when he tries; he "utters," but does not "say." This will remind the reader of what was said of some patients who can reply "no," but cannot say that word when they are told to try. The patient "Dow" uttered the word "damn," one night, when vexed on his daughter coming in very late. Her mother told me of their surprise, and that her daughter said she would stay out late every night to get him to speak. But he could not repeat the expression. I never heard of any utterance in his case but of "damn" on that occasion, and his recurring "no" and "eh" (yes).

As said above, it is not a question of oaths only, but of ejaculations in general (interjections simple or compound).<sup>1</sup> They are all parts of emotional language; their utterance by healthy people is on the physical side a process during which the equilibrium of a *greatly* disturbed nervous system is restored, as are also ordinary emotional manifestations. (All actions are in one sense results of restorations of nervous equilibrium by expenditure of energy.) In some people oaths and vulgar interjection have become very deeply automatic; some people swear largely along with their ordinary unexcited speech, perhaps to give emphasis to commonplaces. In these people the oaths are almost as automatic (their nervous arrangements being strongly organised) as smiles and frowns; they are, so to speak, "detonating commas." No wonder that they are Occasional Utterances when these patients are speechless. Few women swear, but their ejaculations of surprise or vexation (feminine oaths), as "Oh! dear," "Dear me!" "How very tiresome!" belong to the same category. The aphasic woman, whose recurring utterance was "me, me," &c., once ejaculated, "God bless my life!"

<sup>1</sup> I take the following from an unsigned review in the 'Journal of Mental Science' for April 1878, p. 125: "The value of swearing as a safety-valve to the feelings, and substitute for aggressive muscular action, in accordance with the well-known law of the transmutation of forces, is not sufficiently dwelt on. Thus the reflex effect of treading on a man's corn may either be an oath or a blow, seldom both together. The Scotch minister's man had mastered this bit of brain-physiology when he whispered to his master, who was in great distress at things going wrong, 'Wad na an aith relieve ye?'"

It has been said that he who was the first to abuse his fellow-man instead of knocking out his brains without a word, laid thereby the basis of civilisation.

(2) There are occasional utterances which are real speech, but inferior speech. This remark may be indefinite, but illustrations will show what is meant.

I saw, in consultation, a patient who had the recurring utterances "no" and "what." This patient was heard by his doctor to say "Wo, wo!" when standing by a horse. This patient once uttered "That's a lie," which is an expression often used by vulgar people as a verbal missile, that is emotionally rather than propositionally; it therefore comes under No. 1. The utterance "wo, wo!" is the one I wish to draw attention to now. "Wo, wo!" is a proposition to those who use it, if not to animals; it means "stand still."

A woman who could only utter the phrase "Yes, but you know," once said "Take care!" when a child was in danger of falling. A patient of Trousseau's said "*merci*," when a lady picked up his handkerchief. A patient of mine would, besides swearing when vexed (No. 1), say "Good-bye," when a friend was leaving him.

The man who said "Wo, wo!" could not repeat it; and the lady could not repeat "Take care!" The friends of Trousseau's patient thought he was beginning to speak; but he could not repeat the word. My patient could never say "Good-bye," except under the appropriate circumstance; his daughter had found this out herself.

The following is from a communication made to the *Lancet*, May 18, 1878, on this patient's case. Several different phenomena are mentioned; they are all of the same order, in so far that they show conservation of automatic with loss of voluntary action.

"I have seen a patient who usually sat up in his room, whose face looked intelligent, who was cheerful and merry, and who seemed to understand all that I said to him, but who could not put out his tongue when he tried. His daughter remarked that he could put the tongue out, as she expressed it, 'by accident,' and added, as an illustration of her meaning, that when any one was leaving him he could say 'good-bye,' but that he could neither put out his tongue nor say 'good-bye' when he tried. He could say 'yes' and 'no' at any time; and, using the lady's expression, could say 'good-bye,' 'well,' 'never,' *by accident*.

She further remarked that the patient would sometimes swear. He uttered the short explosive word which is so much in favour with English swearers, but he could not, she said, repeat the word when he tried. She asked him to utter the explosive sound when I was there, saying it herself for him to imitate. He laughed, and shook his head."

Admitting the utterances (No. 2) to be exceptions, we have to note that, as exceptions, they are significant. They are true speech, but they are inferior speech. Superiority in speech does not mean number of words, nor even solely precision of application, but precision of application to new relations of things, that is, in effect superior speech is accurate speech on complex<sup>1</sup> matters. We do not find that the loquacious person speaks precisely, except on the most familiar things; on novel things he fails greatly. The "faculty" of speech is not, as popularly supposed, highly developed in him. The utterances are well organised; they were prompted—to speak popularly, helped out—by their special circumstances. They are only in degree less significant than the Occasional Utterances (1), or than the Recurring Utterances (4). Besides this, the inability to repeat them is to be carefully borne in mind.

(3) I have records of still higher degrees of utterance by one speechless patient. A man, for several months under my care in the London Hospital, was absolutely speechless. He never *uttered*, much less spoke, anything but "pooh," "pooh," so far as I or the students or the nurses knew. But I was told by his friends of three utterances. Once, when he had had enough bread-and-butter, he said "No more." This, however, is only a degree of speech on a level with those in the just-given illustrations (2). But I was told that one day the patient said,

<sup>1</sup> Although of necessity we take type cases, we not only consider what we call exceptions to the type, but in actual practice we consider individual peculiarities. What is well organised in one person is not so in another; when we say that the more automatic, more organised, &c., remains, we mean what is more organised in this or that patient. The qualifications to be understood in using such expressions as "*the concept*," "*the English language*," "*the environment*," need not be pointed out. In such expressions as "*from the special to the general*," "*from the complex to the simple*," the obvious qualifications must be kept vividly in mind. When we speak of complexity of any actions, we do not mean any sort of abstract complexity. A man in delirium goes through very complex manipulations of his trade, but they are not complex *to him*.

with difficulty of articulation, "How is Alice [his daughter] getting on?" A third utterance was, I think, as high, if not still higher, in speech. His son wanted to know where his father's tools were. In reply to his son's questions, the patient said, "Master's." Although here is but one word, where in health there would have been a sentence, there is a proposition; it told his son where the tools were as fully as the most elaborately worded and grammatically complete sentence would have done. It was far higher than the most elaborate oaths, and higher even than such utterances as "no more," "good-bye," "very well," &c. Once more I would urge that speciality in speech ("high speech") is not simply an affair of number of words, nor simply of complexity of their arrangement. We have to consider precise adaptation to special and new circumstances: "master's" did not come out upon a common and simple occasion, like "good-bye;" it was definitely uttered to signify a very special relation, moreover a new relation. Granting, for the sake of argument, what, however, I do not know, that the man had in health replied scores of times to the same question by that word, or by a fuller proposition containing it, it was specially used for a new occasion, under, that is, very new circumstances. The father had left his work, would never return to it; was away from home; his son was on a visit, and the question was directly put to the patient. Any one who saw the abject poverty in which the poor man's family lived would admit that these tools were of immense value to them. Hence we have to consider, as regards this and the other occasional utterances, the strength of the accompanying emotional state. We shall consider the influence of strong emotions, which imply great nervous tension on the production of these utterances, later on.

I used to receive reports of these utterances and sayings by speechless patients with great incredulity, and so I find have others done. One of my most intelligent pupils, to whom I was speaking of such utterances, told me that the wife of a speechless patient, with much indignation at his (the student's) incredulity, affirmed that her husband (otherwise speechless since his attack) uttered before he died, "God bless you, my dear!" This utterance, if largely emotional, was a painfully

appropriate one from a dying man to his wife. He may have uttered it interjectionally scores of times when well, and have *said* it when dying; really *meaning* that God should bless his wife. Under some circumstances truths that have died down into truisms become alive again.

The Communist orator who began his oration by "Thank God, I am an Atheist!" used "thank God!" as a mere expletive: even when this phrase is uttered devoutly, it is often more emotional than propositional; but in some states of mind it doubtless is *said* with full propositional intent. Much poetry, in prose and verse, nowadays seems to be an attempt to show the truths of what have become uninfluential truisms.

These utterances naturally surprise the friends of speechless patients. A patient, fatally ill, unable to tell what she wanted (this patient had not entirely lost speech), surprised her sister by exclaiming, "Surely you must know what I mean?" after that she said nothing intelligible. A patient under my observation in the London Hospital could utter many words, but his oaths and other ejaculations were alone properly uttered; a patient in the next bed felt insulted on being asked to note what the patient uttered. Naturally he would feel that a man who, when asked to write, ejaculated, "What's all this bloody nonsense about?" could talk if he liked.<sup>1</sup>

Gairdner had an aphasic under his care in hospital, and wishing to learn something as to the patient's general condition, asked another man in the ward what *he* thought of him. "I think a guid whuppin wad be the cure of him." On Gairdner remarking that the patient could not speak, the man replied, "Na, but he swears whiles;" evidently believing that the poor

<sup>1</sup> Dow's wife told me that the neighbours were very unkind; they said it was all nonsense about his being unable to talk, for why did he not write? They could not be expected to know that if speech goes, writing goes—expression in writing is meant; Dow copied a good deal, and could sign his name without copy. Had they heard Dow utter "Damn," because his daughter came in late, they could have felt the correctness of their opinion of his case to be demonstrated. In general the laity cannot be expected to know that swearing, &c., may persist when speech proper is impossible, and certainly not that a higher kind of utterance may persist when the patient is fatally ill. No doubt many apoplectic persons found in the streets are locked up for drunkenness because the policeman does not know that swearing is a very automatic process, which can persist under conditions produced by fatal brain lesions as well as by drink.



fellow was shamming. The aphasic died, and cancer of the brain was found at the necropsy.

In some cases of speechlessness an elaborate utterance comes out of which we cannot guess the meaning. The following case is an illustration of this and also of other utterances. A patient under the care of Dr. Martin,<sup>1</sup> in St. Bartholomew's Hospital, could only utter the word "yes." The Sister of the ward (a very intelligent lady) remarked that he uttered this word when he meant "no;" moreover, she said he often nodded when he meant "no." One of the nurses told her that the patient once, in words, asked for beer; but the Sister remarked, "I don't believe this, as I was constantly with him, and never never heard him say anything." I think it very likely he did, under strong excitement (active desire), get out a proposition to that effect. It may be said that there could be no excitement about so small a matter. But it is no small matter to many hospital patients. Some will leave the hospital if they do not get beer. But the utterance I wish to draw attention to is the following. His wife said that all she ever heard him utter beyond "yes," was "Five nights, six nights, seven nights, and then five nights out of seven." What this meant she could not guess.

To resume once more. There are three exceptions to the statement that our "Speechless" man is absolutely speechless. He may have permanently the utterance of the words "yes" and "no," and the full use of them: their use as speech. On the other hand, we have noted that a patient may have only the emotional or interjectional use of them, and that when he has, more than this, the ability to reply with them, he may be unable to say them when told. And where there is the full use of them, we have to bear in mind that they are the most general of all propositions. Then he has *occasionally* some inferior speech, and as I believe this to be effected by the right half of his brain, I admit that these occasional utterances show, as do "yes" and "no," some power of speech during activity of that side. Anyhow, they show that the patient retains organisations for some words somewhere in his nervous system.

<sup>1</sup> Dr. Martin kindly allowed me to see this patient and to report it, 'Lond. Hosp. Reports,' vol. iv. p. 365.

There is no demonstration by these cases that the patient retains organisations for any other words than those he actually utters. But is it a likely thing that Trousseau's patient, who said "*merci*," when a lady picked up his handkerchief, had just that word or a few such words left? A fire occurred in the street opposite one of my wards in the London Hospital: a speechless patient of mine cried out "fire!" Is it not a grotesque supposition that this woman retained only the word "fire"? Moreover, those who say a patient tried to repeat any of his occasional utterances are tacitly admitting that the words of those utterances are revived in him; otherwise the word tried has no meaning. There is demonstration by other means that the speechless patient retains a full service of words; he understands what we say to him. At any rate, the utterances spoken about show that there is retention of some words, if only a fragment or so in each case. Some of them also show that there is not only retention of some words, but of some speech, by the right side of the brain. The division we made (vol. i. p. 319) was not that the left half of the brain serves in speech, and the right in receiving speech and in other ways, but that "nervous arrangements for words used in speech *lie chiefly* in the left half of the brain," and "that the nervous arrangements for words used in understanding speech (and in other ways) lie in the right also." It is believed that the process of verbalising and every other process is dual, but that the more automatic a process is, or becomes by repetition, the more equally and fully is it represented doubly in each half of the brain. But the utterances show too, for the most part, that the speech possible by the right side of the brain is inferior speech. In nearly all cases it was well organised automatic or "old," and nearly every utterance required a special occasion, was, to speak popularly, surprised out of the patient by a sudden accustomed stimulus. And it is to be borne in mind that the patient cannot repeat, say voluntarily, what he thus utters. So far these exceptions are exceptions proving the rule.

It has been admitted, however, that occasionally there is an utterance of high speech-value. This exception will be considered after a while.

## VOMITING IN CONNECTION WITH CEREBRAL DISEASE.

BY D. FERRIER, M.D., F.R.S.

THE conditions under which vomiting occurs are so numerous and so diverse that it is difficult to understand how the same mechanism can be set in action in so many different ways. In attempting to explain the various symptoms met with in connection with cerebral disease, vomiting is a symptom which demands special attention, on account of its frequency and grave significance. If the present state of our knowledge does not supply us with data for a definitive solution of the problem, it may, however, be useful to inquire whether we can assimilate vomiting from cerebral affections with vomiting due to other causes, apparently more simple, because more familiar.

Like all reflex actions, vomiting must be looked upon primarily as an indication of adaptive reaction on the part of the organism. The only condition, however, in which this adaptation is at once clearly evident is in presence of morbid irritation of the stomach or upper part of the alimentary canal—vomiting being a reflex reaction adapted for the expulsion of substances causing such irritation. Though no irritating substances should be actually present, yet it is physiologically the same thing, if similar irritation should exist from any cause. Hence vomiting from morbid irritation of the stomach, however produced, is readily explicable.

Without actual irritation of the fauces, or of the stomach, however, vomiting may be caused by unpleasant odours or tastes.

Whether there is any close anatomical relationship between these nerves and the sensory nerves of the stomach, or whether

the solidarity subsisting between smells, tastes, and substances congenial to the stomach is merely the product of association, is a question which might be of interest to discuss; but without entering on this, we may take it as a fact that certain smells and tastes are practically the same thing as gastric irritants.—the senses of smell and taste being merely the advanced guard of the stomach. The connection, it is true, may be broken through by effort and habit, yet this does not invalidate the general rule.

Under the same category as actual unpleasant odours or tastes may be placed the vivid realisation in idea of the same.

For the ideal smell or taste is merely the central reproduction of precisely the same kind and quality of impression, as result from the actual application, at the periphery of the olfactory or gustatory apparatus, of the evil-smelling and evil-tasting substances. All the other physical concomitants tend likewise to be reproduced, and hence vomiting may occur if the ideal reproduction is sufficiently vivid.

Closely allied to vomiting from such causes is the occurrence of vomiting from the feeling of emotion or Disgust.

The facial expression of disgust coincides with the natural reflex movements of the nose and mouth indicative of repulsion of unpleasant odours or tastes. Disgust and its facial expression are no doubt primarily founded on objects repulsive to taste and smell, and therefore unfit for food; but by association, various other secondary objects have been engrafted on to it, related at a greater or less distance with things abhorrent to the stomach.

Hence disgust can be excited and vomiting actually produced by such things as actual or ideal contact with cold slimy things, or with filth, putridity, or messes in general. Disgust reaches its acme by processes of sympathetic realisation when we see others dealing with things the very sight of which is repulsive to us. The sight of vomited matters, and still more the sight of a person vomiting, especially if there is any tendency to sickness already existing, are sufficient to bring matters to a crisis.

In all these cases there is actual irritation, conditioned either

centrally or peripherically, of the sensory nerves of the stomach, or their physiological associates.

Though vomiting is specially serviceable in expelling irritants from the stomach and upper part of the alimentary canal, yet, as we see in stercoraceous vomiting, it may prove efficient in emptying the intestinal canal. Hence the occurrence of vomiting in connection with irritation of the alimentary canal, otherwise than in the stomach, may be brought under the same law of adaptive reaction. Vomiting therefore in connection with strangulated hernia would thus be accounted for.

But vomiting from direct irritation of the alimentary canal forms only a small proportion of the conditions under which it may be manifested. Vomiting is of frequent occurrence in connection with irritation, variously induced, of the abdominal and pelvic viscera. We find it with diseases of the liver, more especially during the passage of a biliary calculus; with inflammation of the kidney, and specially during the passage of a renal calculus; with diseases of the spleen, and pancreas; and in a very marked degree in connection with irritation of the uterus and ovaries. Irritation of the peritoneum also, and of the omentum, as in cases of omental hernia, are frequently associated with vomiting.

In vomiting so caused it is not easy to see any such adaptiveness as is clearly manifest in relation with direct irritation of the stomach or alimentary canal. A closer examination, however, may discover in vomiting under some of these conditions a reaction possibly beneficial to the organism.

Thus in the case of an impacted biliary calculus, the violent compression and concussion of the abdominal viscera during the act of vomiting may expedite its passage and remove the source of irritation. Similarly also in the case of a renal calculus. And that the uterus may in this way expel an irritant from its interior, such as retained menstrual fluid, is a fact of which it would be easy to give examples. But vomiting due to irritation of the abdominal or pelvic viscera and their investments, is just as often, if not more so, hurtful than beneficial. So that if we were to look upon vomiting as in all cases the indication of a *vis medicatrix naturæ*, we should have

to admit that this is capable of making gross and pernicious blunders.

In vomiting so induced, we may see rather an illustration of the laws of irradiation which have been established in reference to spinal reflex actions. The reflex action consequent on irritation of any particular spot is at first more or less definite, and limited in reference to the source of irritation; but if the irritation is stronger and more continued, or if the reflex excitability is heightened, there is a tendency to irradiation and the discharge of other movements. This irradiation follows certain definite lines; those movements being first discharged which are most closely allied and symmetrical than others at a greater distance, until ultimately a general convulsion may result.

Though coughing is primarily adapted for the expulsion of irritants of the air-passages, and is conditioned mainly by irritation of the sensory nerves of the lungs, yet it may be set in action by irritation of closely related parts, as by irritation of the pleura, in which case no useful purpose can be effected, but rather the reverse. Similarly sneezing, though primarily conditioned by irritation of the nasal passages, may be excited by a bright light, apparently by mere contiguity.

The anatomical conditions of the innervation of the viscera by closely-connected plexuses of the vagus and sympathetic nerves are such as to render irradiation of impressions from one point to another more than usually easy. Numerous channels are open for the transference of impressions; hence *Synæsthesiæ* or sympathetic sensory neuroses are common, and the same facility exists for the excitation of *synkineses*, or sympathetic motor reactions. The nearest reflex reaction is the act of vomiting, but coughing may also occur, or, if the irritation is great, general convulsions may be induced.

We may therefore regard vomiting in connection with visceral irritation mainly as an indication of irradiation, and the excitation indirectly of the reflex reaction which is specially characteristic of irritation of the stomach and alimentary canal.

But when the irritation is of an intense character, another factor has to be taken into account in addition to the mere

fact of irradiation, viz. the sensation of pain. For intense pain may of itself cause vomiting, apart from any particular seat. As a general rule, however, vomiting occurs most frequently in connection with intense pain in organs and parts whose sensibility under ordinary conditions does not distinctly form part of our consciousness.

Owing to the intense pain which accompanies the passage of a biliary or renal calculus, vomiting here is not a simple case of irradiation. But that vomiting may result from irradiation of visceral irritation not involving pain is evident from the vomiting of pregnancy due to uterine irritation. Vomiting caused by pain leads to the consideration of another class of conditions in which vomiting is frequently seen, independently of direct irritation of the alimentary canal or indirect irritation by irradiation. Vomiting is common in concussion of the brain and in shock from severe traumatic injuries. Concussion of the brain, and shock with syncope are essentially the same. The main features in both are a temporary annihilation of consciousness, with a more or less enduring state of the system in which intense depression of the circulation is the most marked phenomenon. There is great muscular prostration, the face is pale, the skin cold and covered with a clammy perspiration, the temperature is lessened, the pulse almost imperceptible, and the vascular tension almost nil. In this state there are frequent attempts at vomiting or actual sickness.

Shock has been shown by Dr. Lauder Brunton ("The Pathology and Treatment of Shock and Syncope"—'Practitioner' vol. xi. p. 241) to depend chiefly on dilatation of the abdominal blood-vessels.

The most efficient cause of shock is traumatic injury of the abdominal viscera. A smart tap on the intestines of the frog, as in Goltz's experiment, almost annihilates the circulation by causing reflex dilatation of the abdominal vessels. The abdominal vascular area is so great that when fully dilated the abdominal vessels can accommodate nearly the whole of the blood of the system. In such case, even though the heart may continue to beat, little or no blood flows through it, and therefore the circulation is only not at a complete standstill.

The same cause also usually induces reflex stoppage of the heart, and thus we have shock combined with syncope or temporary annihilation of consciousness, but the syncope may pass off and the symptoms characteristic of shock continue.

The accumulation of blood in the abdominal viscera is sufficient to account for the pallor of the skin and the other phenomena indicated, for though the other vessels might also be dilated, yet they would be drained of their contents.

The phenomena of syncope and shock would seem to show that vomiting may be induced by conditions causing great lowering of the blood-pressure and depression of the heart's action.

This is further borne out by the effects of copious hæmorrhage. Venesection *ad deliquium animi* was a frequent cause of sickness and vomiting.

The substances which act most powerfully as emetics, apart from those which have a directly irritant effect on the stomach, have an intensely depressant action on the circulation, and produce all the phenomena characteristic of shock. Vomiting is almost an invariable accompaniment of the toxic effects of the cardiac poisons. Some of these, like veratria, may also exercise a directly irritant action on the alimentary canal, but in others the vomiting is out of all proportion to the irritant effects which are discoverable.

From these considerations it would appear that whatever causes great depression of the circulation may cause sickness and vomiting, and thus we may bring into relation with each other conditions which at first sight seem to have nothing in common. Vomiting, as has been observed, is frequently seen in connection with great pain. A blow on the testicle, a loose cartilage suddenly coming between the articular surfaces, a dislocation, a traumatic injury of the eyeball, and many other forms of pain have been known to cause nausea and vomiting.

The experiments of Mantegazza (Schmidt's 'Jahrbücher,' 1867, 133) show that intense pain causes a depression of the circulation similar to what is seen in shock and syncope; and Weir Mitchell has related a number of cases ('Injuries of Nerves,' p. 138) in which shock resulted from gunshot wounds of nerves. From these latter it would appear, however, that



the shock is not in proportion to the consciousness of pain, but to the physiological reflex effects of the injury itself.

The conditions which give rise to the consciousness of intense pain are such as to induce the phenomena characteristic of shock in a greater or less degree, and therefore also nausea and vomiting. The intense depression of the circulation is the common point of agreement. The physical effects of intense mental pain, such as is exhibited in the emotions of fear and terror, are precisely the same as those of intense bodily pain, and act through the same channels. Hence nausea and vomiting may result under essentially the same conditions. Suffering in others has often a sickening effect on bystanders. This is frequently met with in those who witness a surgical operation for the first time. The suffering of the patient, or the supposed suffering, is realised sympathetically by the onlooker.

In addition to these various causes of nausea and sickness there is another condition in which these symptoms are specially apt to occur, viz. in connection with vertigo and disorders of equilibration. The sickness may in part be due to the terror which arises from a sudden sense of insecurity, yet there are many considerations which point to a direct relation between the viscera and the centres of equilibration.

Thus visceral disturbances are very often associated with vertigo. And as visceral disturbances, mechanical or otherwise, induce vertigo, so the motor inco-ordination, of which the sense of giddiness or vertigo is only the subjective accompaniment, whether due to central causes or to peripheral affections, as in labyrinthine vertigo, tends, by a process similar to irradiation, to produce visceral commotion, and with it nausea and vomiting.

As regards the immediate physiological antecedents of vomiting in connection with great depression of the circulation, we can say nothing very definite as to whether there is direct irritation of the vagus roots in the so-called vomiting centre, or indirectly from the condition of the heart and blood-vessels, of which the abdominal blood-vessels are the most important.

But that irritation of the vagus, centrally or peripherically, is the ultimate fact may be fairly assumed, and thus we may

see a closer relation between vomiting caused by irritation of the stomach and alimentary canal, and vomiting under the various conditions described than seems at first sight to exist.

It is possible also to see in vomiting in connection with depression of the circulation a reaction distinctly beneficial to the organism.

Thus in cases of vascular dilatation in the splanchnic area, the violent compression and concussion of the abdominal walls in the act of vomiting serve to propel the blood onwards, and thus raise the blood-pressure. It is observed in concussion of the brain and shock that vomiting is generally the precursor of recovery, the blood-pressure rising and the circulation re-establishing itself. Marshall Hall also, in describing the effects of loss of blood, calls attention to the fact that "the state of syncope is often relieved, for a time, by an attack of sickness and vomiting, immediately after which the patient expresses himself as feeling better, and the countenance is somewhat improved, the breathing more natural, and the pulse stronger and more frequent" ('On the Effects of Loss of Blood,' p. 11).

With this brief review of the chief conditions under which vomiting is met with, apart from affections of the brain, we may now inquire in what relation, if any, cerebral vomiting stands to them.

Many grave organic diseases of the brain run their course without vomiting as a symptom. The forms of cerebral disease in which vomiting is especially met with will be found on examination to correspond with those in which headache is also a prominent symptom, and therefore, as I have previously ('BRAIN,' Part V.) endeavoured to show, in cases where there is irritation of the nerves of the cerebral membranes by inflammation, excessive tension, and the like.

Hence vomiting is especially manifested in connection with meningitis and cerebral tumours. Vomiting occurs in affections of this kind altogether independently of the position of the lesion. There is no evidence of any value in support of Budge's assertion that vomiting is more frequent with disease of the right hemisphere than the left. Nor does there seem better evidence for his view that the right optic thalamus and

corpus striatum have special relation to the stomach, than his view that the cerebellum has special relation with the testicles.

There is no evidence, physiological or pathological, that direct irritation of the substance of the brain, apart from irritation of the cerebral membranes or other complications to be mentioned, is capable of exciting vomiting. Vomiting and headache generally go together.

It would not, however, be correct to assert that vomiting in cerebral disease is proportional only to the intensity of the pain. That the intensity of the pain alone, particularly of the agonising character frequently seen in cerebral tumours, may be sufficient to cause sickness and vomiting, would be in accordance with the effects of intense pain in other regions, and would be explicable in the same way.

But we may observe vomiting of a pathognomonic character in the early stages of tubercular meningitis, before pain has reached any marked degree of intensity. The headache and vomiting do not stand in direct relation to each other.

Frequently, in fact, pain in the head and vomiting alternate with each other, the headache giving way to vomiting, and *vice versâ*.

We have therefore reasons for concluding that vomiting may be excited by a degree of irritation short of that necessary to cause intense pain with its constitutional depression. In fact the comparative ease of cerebral vomiting without the marked nausea and depression which accompany sickness from intense pain, would seem to indicate a specially close relation between the sensory nerves of the cerebral membranes and the centres concerned in vomiting, the resemblance between cerebral vomiting and uterine vomiting being closer than between the former and vomiting from a renal or biliary calculus.

The facility of irradiation of visceral irritation and the excitation of vomiting has been already alluded to, but the question is whether there is such an intimate association between the sensory nerves of the cerebral membranes and those of the stomach as to account for cerebral vomiting by a similar process of irradiation.

As regards the physiological association, we have sufficient evidence in the connection between headache and gastric disturbances, so constant that the head is in a measure the sensory index of the stomach. This in all probability is to be explained by the close anatomical relationship between the nuclei of the vagus and trigeminus in the medulla oblongata, rendering the transference of impressions from one nerve to the other comparatively easy. Hence vomiting from irritation of the cerebral membranes may be brought under the same principle of irradiation as vomiting from visceral irritation not directly affecting the stomach. As in this case, so in irritation of the cerebral membranes, there is a synæsthesia of the stomach, and vomiting in consequence. This mutual synæsthetic relationship between the nerves of the stomach and cerebral membranes seems to me to account most satisfactorily for the phenomena of migraine or sick headache. Here we have an association of intense headache and vomiting, the two symptoms often alternating with each other, and also very frequently certain remarkable paræsthesiæ or even parakineses, chiefly unilateral, which may be accounted for by concomitant affection of the cortical centres in immediate relation with the seat of irritation in the cerebral membranes. The clinical history and etiology of migraine accord well with the hypothesis that it is essentially a neurosis of the cerebral membranes.

While we may ascribe the great majority of cases of cerebral vomiting to irradiation of irritation of the nerves of the cerebral membranes, or to the physical effects of acute pain, there are some cerebral affections in which possibly another cause may be operative. It is generally believed that vomiting is more especially associated with lesions of the cerebellum and corpora quadrigemina. Diseases affecting the centres of equilibration might be accompanied by sickness more through the vertigo induced, than from mere irritation of the cerebral membranes.

In the facts recorded, however, it is not easy to eliminate what may be due to the lesion as such, and the causes operative here as elsewhere. For the anatomical relations of the posterior fossa of the skull are such as to allow of irrita-

tion of the cerebral membranes often of a very definite and circumscribed character. Should vomiting be proved to be present in such cases apart from irritation of the membranes, we might account for it by disturbances of equilibration and the concurrent vertiginous sensations. But, apart from these circumstances, irritation of the cerebral membranes seems to afford a sufficient explanation of most cases of cerebral vomiting.

## Critical Digests and Notices of Books.

*Luciani and Tamburini on the Functions of the Brain. The Psycho-sensory Cortical Centres.* 'Sulle Funzioni del Cervello. Ricerche Sperimentali dei Professori Luigi Luciani e Augusto Tamburini. Seconda Comunicazione: Centri Psico-Sensori Corticali. 8vo. pp. 78. Reggio—Emilia. 1879.

IN this communication the authors confine themselves to an experimental inquiry into the centres for visual and auditory perceptions. The method of investigation consists, as before, in exposing, under chloroform, certain convolutions by the trephine, examining the electric excitability, and thereafter carefully removing the cortex so as to avoid hæmorrhage as much as possible.

I. In their historical introduction, the authors mention that Flourens, the founder (1842) of the scientific inquiry into cerebral localisation, held that the brain is an organ, physiologically one, performing all its functions both as a whole and in every part. According to his view, therefore, partial mutilations of the brain would weaken the power of performing all functions, and when complete or great removal was effected, all kinds of psychical activity would be annihilated. Hearing and sight would therefore disappear on removal of the mass of the brain. This view was combated by many, and Schiff contended (1858) that not even removal of a complete half of the cerebrum had any influence on vision. Meynert (1866) by following the course and termination of the hemispherical fibres of the auditory nerve, came to the conclusion that to the cortical substance of the convolutions of the fossa of Silvius belonged the function of receiving impressions of sound.

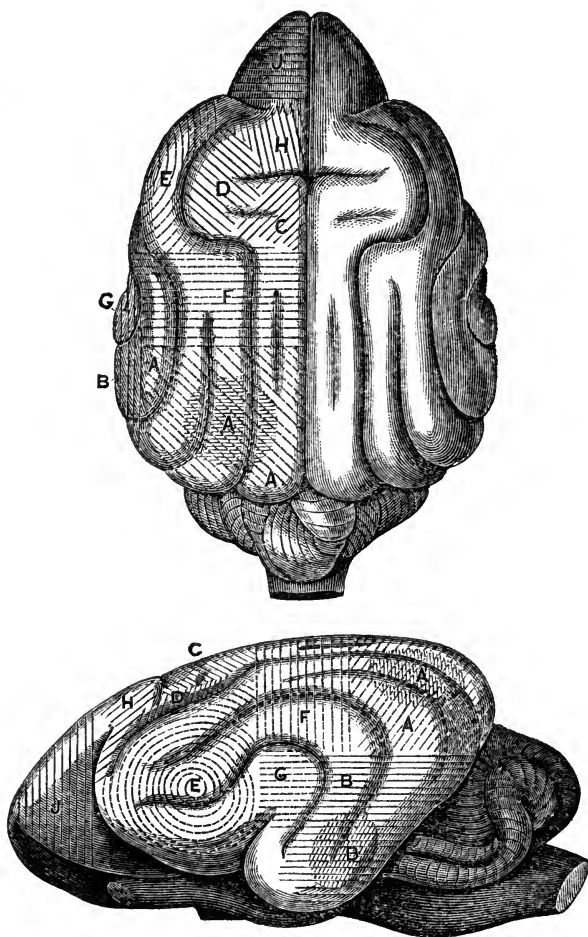
Charcot has recently said that the cortical sensory centres would probably be found in the temporo-sphenoidal and occipital lobes. In 1874 Hitzig first pointed out briefly that lesion of the posterior lobe of the brain can produce blindness of the opposite eye.

By means of the water jet, Goltz showed that when an important part of the cerebral cortex is removed, temporary blindness of the opposite eye ensues. This result follows (he says) even after removal of the so-called motor zone of Hitzig. Although the animal recovers, it does not do so completely, the animal never quite recovering the power of appreciating distance and of distinguishing the nature of objects, and being impassive. He does not deny that disturbance of other senses than sight (hearing, for instance) may ensue from such a lesion, but does not say he has completely investigated the matter.

Ferrier showed that a much larger portion of the cerebral cortex than Hitzig had succeeded with was excitable, and he attempted to localise (among other sensations) hearing in the superior temporo-sphenoidal convolution, and sight in the angular gyrus, or *pli courbe* of monkeys. In dogs, the visual centre, according to Ferrier, is located in the parietal portion of the second external convolution, while the centre for hearing is defined as the upper and posterior portion of the third external convolution. Ferrier confirms his view of these localisations, drawn in the first instance from the occurrence of certain (motor) reactions on irritation of the centres, by proceeding thereafter to remove the centres. He maintains that unilateral removal of the angular gyrus is followed by complete, but not permanent, blindness of the opposite eye, while after bilateral destruction it is complete and permanent. In the case of dogs and cats, Ferrier's experiments are less complete, and he quotes only one experiment where he destroyed the parietal portion of the second external convolution in a cat, with the apparent result of complete blindness in the opposite eye. He believes that the temporo-sphenoidal lobes in monkeys, and the upper and posterior part of the third external convolution in dogs, are centres of *auditory sensations*, and believes that removal of these centres is followed by annihila-

tion of the sense of hearing, though admitting the difficulty of settling the question.

Hermann Munk has experimented since Ferrier, and does not altogether agree with him.



Functional localisations of the cerebral cortex of the dog, according to Munk.

- A. Visual sphere. B. Auditory sphere. C. Sphere of the posterior limbs.  
D. Sphere of the anterior limbs. E. Sphere of the head. F. Sphere of the eyes.  
G. Sphere of the ears. H. Sphere of the neck. I. Sphere of the trunk.

According to this writer, the brain of dogs may be divided into an anterior or motor portion, and a posterior or sensory,



by a vertical line passing from the end of the fissure of Silvius to the falx. Destruction of the centres in front of this line induces lesions of motility, while removal of those behind it does not produce any motor phenomena. If the lesion affects the occipital lobe towards its posterior superior extremity, (A, Fig. p. 236) certain characteristic disorders of the visual function ensued, which Munk explains as the effect of amnesia of the visual images, and which he terms *psychic blindness*; when, on the other hand, it affects the temporal lobe near its inferior extremity (B, Fig. p. 236) special changes of the auditory functions are manifested, which he explains as effects of (amnesia) loss of memory of auditory images, and terms *psychic deafness*. If the ablations do not involve the said segments, but other areas placed just in front of and below them, limited disorders follow which last only three to five days, and which Munk entirely overlooked in his first researches.

The memory of single visual (and presumably also of auditory) images would, according to Munk, have a fixed and determined site in the cerebral cortex. He believes, in fact, that he has shown that a dog on one occasion lost memory of all visual images, except that of the dish from which it was accustomed to drink; and that another dog remembered only the gesture in response to which it had been previously accustomed to extend its paw. His explanation is, that in the case of the first, all the parts of the cortex had been removed except that part which retained the memory of the dish; and in that of the second, all those parts except that which retained the memory of the manual gesture. Munk asserts further that the forgetfulness of visual and auditory images passes off, and in four to six weeks the animals experimented on cannot be distinguished from those perfectly sound. He compares such animals with those newly born, and says that, like these, they gradually lay up ideal images of auditory and visual sensations. The question then arises, Where are such representations deposited, since the physical substratum naturally set aside for them has been removed? Munk says, in the parts of the brain surrounding those parts. He has not been able definitely to localise the new representations, however, because all his animals died of most acute meningitis in his efforts to remove

the surrounding centres. So also did all the animals from whom he removed the visual centres plus a radius of twenty millimetres surrounding them, in order to determine when the normal function failed to be restored. In the case of two dogs, he succeeded in removing the whole cortex of the left hemisphere of the occipital lobe (A A A). This was followed by complete blindness (not simple psychic blindness) of the right eye, which passed off after some time. He could not clearly determine whether the mnemonic images returned; and when he tried to remove the corresponding portion of the right occipital lobes, both the dogs died.

In monkeys, according to Munk, the visual sphere is localised in the cortex of the occipital lobes, and not in the angular gyrus as maintained by Ferrier. The extirpation of a circular area (10-15 mm. in diameter) in the occipital lobe produces limited disturbances of vision; the animal seems to have lost the memory of visual images for certain objects, preserving those of others. If the entire cortex of the convex surface of the occipital lobe is destroyed, the animal becomes hemiopic or cortically blind in the halves of both retinae on the same side as the lesion, on which half of the retinae the animal does not recognise objects, though it does so perfectly on the opposite half. If the cortex of both occipital lobes is removed, the monkey becomes entirely blind, habitually quiet and apathetic, and when compelled to move, it stumbles against every obstacle in its path. After a certain time, vision partially returns, so that the animal can walk about slowly without stumbling. If certain portions of the cortex at the margin of the convexity of the occipital lobe remain intact, a greater amount of recovery may occur, and hemiopia in the halves of the retinae corresponding with the less completely destroyed side can be made out. Monkeys are essentially, therefore, different from dogs, in whom Munk has never been able to make out bilateral visual disorder after unilateral lesion.

Munk's latest view of the motor region of the cortex has been harmonised with the view just given of the visual and auditory functions. He looks upon what is generally considered the Motor or Psycho-motor region as sensory in the widest sense, or as the seat of the sensory perceptions of the body, and

of the images or sensory concepts which are thence derived. Destruction, therefore, of this sphere produces psychic paralysis of the said perceptions, and loss or forgetfulness of the sensory images there resident, partial or complete, according to the amount removed. Motor disorders on this view are the results pure and simple of abolished cutaneous muscular and nervous sensibility. He makes seven spheres of his sensory region, of which Luciani and Tamburini deal only with two, the sphere of the eyes and of the ears. The ocular sensory sphere, according to Munk, is found in front of the visual sphere, and corresponds in monkeys exactly with the angular gyrus (the visual centre of Ferrier), and in dogs with a zone widely extended transversely, which "comprehends a large median segment of the first, second, and third external convolutions (including, therefore, a portion of the centre for the orbicularis palpebrarum, and of the visual centre of Ferrier).

When ablation of these regions is carefully performed, neither less nor more being removed, the following phenomena are said by Munk to occur. When the opposite eye is pricked or touched, the lids being held open, the animal merely winks, but does not otherwise move. When the eye of the same side is similarly handled, the animal attempts to protect itself and to get away, besides winking violently. The opposite eyelids do not wink on mere manual gesture before the eye without contact. In Munk's view, this implies simple paralysis of the sphincter, as according to him the visual perceptions and conceptions are intact. When the eye of the dog is bandaged, or a stitch put in the eyelids of the monkey on the same side as the lesion, the animals take by mouth or hand respectively small pieces of food offered them. Sometimes there is observed on the side opposite the lesion a slight ptosis and lacrimation, more or less, particularly in the monkey. No difference in the pupils. From these facts Munk infers that these are the special sensory spheres for the eyes.

In front of the auditory sphere, according to Munk, is found the special sensory sphere for the ears, corresponding in dogs with the upper portion of the Silvian or fourth external convolution; and in monkeys with the upper part of the superior temporo-sphenoidal convolution. In the first weeks after uni-

lateral extirpation of these regions he has noticed that the movements forward and backward of the outer ear of the opposite side are absent or weaker than those on the same side. In the dog there also occurs a loss of sensibility, particularly in the convex surface of the opposite pinna.

II. Of the effects of excitation and of destruction of the anterior or frontal portion of the second external convolution of the brain in dogs.

To Goltz belongs the honour of having shown that ablation of the motor zone in dogs is sometimes followed by blindness of the opposite eye. The authors observed this in only one case (Expt. XIX. of their previous communication), and in that case they think it not unlikely that the inflammatory softening, to which they trusted for destruction of the motor zone, had involved also the psychic centre of vision. They point out, however, that Ferrier's area 7.7.7. on the frontal portion of the second external convolution occupies an extent of surface quite disproportionate to the very small motor function which he localises there, namely, *conjoint action of the orbicularis oculi and zygomatics*.<sup>1</sup> In point of fact, they shew that removal of the anterior portion of the second external convolution in dogs is followed by disorder of vision on the opposite side, not amounting quite to blindness, but to a marked obscuring of the perception of visual images, so that objects appear as if covered by a mist. This obscurity passes off after a time. (Expts. II. and III.) And they state it as their opinion that the contraction of the opposite orbicularis oculi which follows irritation of this portion of the cortex should be considered as a reflex effect of subjective sensation, comparable with that which is commonly observed on the sudden appearance of a bright light before one eye. To the question: Where then is located the centre for movement of the orbicularis oculi, if the frontal portion of the second external convolution is partly a visual centre, as they maintain? they cannot give a direct answer. But inasmuch (1) as destruction of this zone is fol-

<sup>1</sup> Ferrier mentions movements of the eyes as also following irritation of this centre; but the authors' statement still holds good, that there seems to be set aside for these simple actions a very large portion of cerebral substance.—Tr.

lowed by ptosis of the opposite orbicularis oculi, and (2) because certain epileptic seizures, which have been proved to depend essentially on a state of tension of the motor zone of the cortex, begin in the orbicularis, they think the motor centre for the orbicularis is probably located in this portion of the cortex. And they think (though without offering proof) that this special centre probably lies well forward near the rest of the motor zone. The relation of the authors' view to that of Ferrier is now sufficiently evident. As to Munk, he places this portion of the cortex in the sensory region of the head, destruction of which would in his view cause psychic paralysis of the opposite half of the tongue and of the muscles of the mouth, and loss of sensibility in the opposite half of the face generally; but not one of his 100 experiments brings out the importance of this segment of the cortex to vision.

III. Of excitation of the median or parietal portion of the second external convolution in the brain of dogs and cats.

According to Ferrier, excitement of this part induces the directing of the eyes to the opposite side; and in the experiments of the authors (IV., V., VI., VII., VIII., IX.) there is substantially the same effect, though once (Expt. VII.) there was seen movement of the opposite eye to the *same* side. But this happened after an epileptic seizure.

They say, however, that they have localised more definitely than Ferrier the effects as regards the action of the orbicularis and palpebral elevator. He says that the effect of irritation of this region is contraction of the orbicularis, tending to close the eye; though if the eye was closed, to begin with, it opens slightly at the moment of passing the current. They say that contraction of the orbicularis (closure of the eye) occurs when they irritate the anterior, and opening of the eye (contraction of the elevator) when they irritate the posterior part of this region. Further they insist, in opposition to Ferrier, that myosis is far from constant; that sometimes it is absent, and sometimes there is midriasis.

They do not think these different results can be explained by different degrees of narcosis, of excitability, and of stimulus

applied, and they come to the conclusion that this part of the brain is not an organ functionally homogeneous. It is, they say, only on ablation of the centres that sound conclusions can be founded, and as Ferrier admits that there are but few exact experimental data to go upon, they have paid the more attention to

IV. The effects of destruction of the median or parietal portion of the second external convolution in dogs and cats.

Expts. X. to XIX. deal with this subject for dogs, and XX. and XXI. for cats, with the result that the parts of the brain in cats homonomous with the same parts in dogs appear to have the same functional value. The following general conclusions are drawn by the authors.

1. Unilateral destruction of the median or parietal portion of the second external convolution of dogs and cats produces immediately amaurosis nearly complete of the eye of the opposite side, and slight amblyopia of the eye of the same side.

2. These disorders are not permanent; but gradually pass off, until compensation apparently perfect is attained.

3. Bilateral destruction of this region produces immediately amaurosis almost complete of both eyes. This appears to pass off indeed, but not so quickly, so that after several weeks some traces of it still remain.

4. This disorder of the visual faculty on both sides is observed even when the bilateral mutilation takes place in two distinct operations, practised at a long interval of time and after compensation had been effected for the disorders consequent on the first operation.

5. These morbid phenomena of visual function take place with nearly the same intensity, when the destruction is limited exclusively to the middle portion of the second convolution, and does not affect parts of the third or first external convolutions.

6. They show on the other hand an intensity and duration notably greater, when the destruction, besides involving the median (parietal) portion, extends more or less also to the anterior (frontal) or posterior (occipital) portion of the second external convolution, and is not limited to the superficial

substance of the cortex, but extends more deeply into the grey matter surrounding the sulci.

V. Of the effects of destruction of the posterior or occipital portion of the second as well as the first external convolution of dogs.

According to the view of the authors it will have been seen that they consider with Ferrier that the median or parietal part of the second external convolution has the greatest importance so far as regards vision. But they assert in contradistinction to him that the centre is not confined to this region but extends forwards, comprehending almost the whole of the anterior part of the same convolution.

Munk, on the other hand, places the visual sphere in the extremity of the occipital lobe, its anterior boundary embracing scarcely any part of the visual region of Ferrier. He places the *sensory sphere for the eyes* in front of his visual sphere. He therefore makes two statements, a negative and a positive. On the one hand, he denies that the cortical visual centre of dogs extends much beyond the boundaries of the occipital lobe; on the other, he states that for the greater part of its extent the occipital lobe represents this centre. The first or negative part of Munk's thesis has been already dealt with by Luciani and Tamburini, who entirely disagree with it. And they take exception to the proof by which he supports it, pointing out that after extirpation of what he calls the sensory sphere of the eyes (in front of his visual sphere), the failure of winking which is noticed when gesticulations are made before the opposite eye, is due rather to interference with vision than, as he maintains, to the fact that the cerebral cortex is no longer competent to put the palpebral sphincter into action.

In order to settle the second part of Munk's thesis, our authors have performed Expts. XXII. to XXV., and have removed the posterior portions of the first and second external convolutions. In three of the four experiments the animals continued to see, they walked freely, could see a lighted taper and follow it with their eyes, and did not stumble against furniture or other obstacles, &c.

In Expt. XXIV. the upper and posterior part of the second external convolution was exposed on both sides in a young dog, to the extent of  $3\frac{1}{2}$  centimetres, by means of an oval opening. The whole region responded to electrification, there being conjugate-deviation of the eyeballs to the side opposite the excitation.

They then destroyed by the sub-meningeal method not only the two portions laid bare, but also the greater part of the occipital lobes, dipping the scoop backwards against the tentorium, and inwards as far as the falx. Hæmorrhage rather copious. Notable consequent depression.

Next day the animal was somewhat depressed. On moving and shaking a lighted taper before the eyes, there was no reaction, or very little; but on threatening the animal with slight blows with the point of the foot, it responded by movements and cries, even when the foot did not reach the snout. The sensibility to pain most acute. Would not move.

During the following six days the animal behaved apathetically. Would not walk, and when compelled to, growled threateningly. Showed active signs of fear, and on being touched, howled lamentably, &c.

Here then was undoubtedly disorder of vision, but the authors believe that the removal of brain substance extended forwards, and involved without doubt a part of the excitable area, where Ferrier localises the visual centre.

While, however, the authors deny that this part of the brain is the visual centre, they do not deny that dogs in whom it has been removed behave differently from normal dogs. They are, for example, apathetic and indifferent to their surroundings; when not running after their food, they tend to remain crouched and inert, seldom responding by winking to gestures made before their eyes; they lose their natural affection, and, if threatened, they appear overcome with fear, and respond rather in defence than offence. This combination of phenomena agrees fairly with those described by Munk as the effect of destruction of the central portion of the visual sphere. But they cannot agree with his explanation when he says, "By the extirpation the dog becomes psychically blind (*seelenblind*), i.e. it has lost the visual representations (*Gesichtsvor-*



*stellungen*) which it had, or the mnemonic images (*Erinnerungsbilder*) of its former visual perceptions (*Gesichtswahrnehmungen*), so that it no longer knows or recognises what it sees; but the dog sees; the visual sensations reach its consciousness and are noted, and allow of the formation of concepts regarding the existence, form, and locality of external objects, by means of which it anew acquires visual concepts, anew mnemonic images of visual perceptions."

The authors put their objection to this view in the form of the following dilemma. "Either the cortical centre in which visual perceptions have their seat is (as Munk assumes) also the centre in which mnemonic representations of previous perceptions are deposited, in which case the loss or oblivion (amnesia) of the representations implies as a consequence loss of the perceptions; or the seat of the mnemonic representations is distinct from the seat of the perceptions, in which case the latter and not the former will represent the true visual centre of the cortex."<sup>1</sup>

VI. Under the head of the effects of electrifying and destroying the parietal portion of the third external convolution in dogs, the authors formulate these conclusions.

1. Unilateral destruction of the upper and posterior part (parietal region) of the third external convolution in dogs produces immediately bilateral deafness, but to a greater degree in the ear of the opposite side.

2. The difference in the auditory sensibility in the two sides constantly diminishes, and after some days disappears altogether; though there are no sound arguments to prove perfect recovery of the auditory faculty.

3. After comparative equalisation of the power of hearing, removal of the corresponding region of the other hemisphere is followed at once by almost absolute deafness, which is approximately equal on both sides.

4. This bilateral cophosis passes off gradually, but data do

<sup>1</sup> I hope on a future occasion to make some reference to what as it stands does not seem to do more than raise the verbal question—What part of the cortex should be defined as the visual centre? Does it not also to some extent imply a misunderstanding of Munk's position?—A. R.

not yet exist for determining whether and when perfect compensation takes place.

5. When removal is confined to the second external convolution, and does not extend to any part of the third, there does not occur the slightest sign of dulness of the auditory sense, which indeed sometimes appears unusually sensitive.

VII. Of the effects of electrifying the angular gyrus in monkeys, as well as the surrounding portions of the cerebral cortex.

The widest divergence of view between Ferrier and Munk appears under the question of the localisation of the visual centre in the cortex of monkeys. Ferrier places it in the angular gyrus or *pli courbe*, while Munk locates it on the convexity of the entire occipital lobe. Ferrier's view is thus expressed: On electrifying the angular gyrus, "The eyes move towards the opposite side, with an *upward* or *downward* deviation, according as the electrodes are on 13 or 13'" (anterior or posterior limb of the *pli courbe*). "The pupils also generally become contracted, and there is a tendency to closure of the eyelids, as if under the stimulus of a strong light. The head frequently follows the direction of the ocular movements, though this is not always observed."

When the superior temporo-sphenoidal convolution is stimulated, there is "pricking of the opposite ear; head and eyes turn to the opposite side; pupils dilate widely."

And according to him the whole cortex of the occipital lobe as also the middle and inferior temporo-sphenoidal lobes are absolutely inexcitable.

The conclusions of the authors are thus expressed:

1. The only one of the phenomena noted by Ferrier as occurring on stimulation of the angular gyrus, which always does occur, is conjugate deviation of the eyes to the side opposite to the one excited. The contemporaneous elevation or depression of the eyes does not constantly follow the rule indicated by Ferrier.

2. The contraction of the pupils noted by Ferrier, although it sometimes appears, is by no means a constant occurrence. Sometimes the pupils undergo no change; but oftener there is midriasis more or less marked.

3. The closure of the eyelids is also a phenomenon of inconstant occurrence, which is not uncommonly replaced by opening of the lids.

4. The occipital lobe, and the upper portion of the middle temporo-sphenoidal, which Ferrier declares to be quite inexcitable by the electric stimulus, appear, on the contrary, in our experiments, capable of reactions perfectly similar to those which arise on excitation of the angular gyrus, only less conspicuous. This is the most important difference between the assertions of Ferrier and the results obtained in our experience.

VIII. Of the effects of decortication of the angular gyrus, as well as the occipital lobe, and of the superior temporo-sphenoidal lobe in monkeys.

Ferrier's view is given in these words: "Destruction of the angular gyrus on one side causes blindness in the opposite eye. The loss of vision is complete, but is not permanent if the angular gyrus of the opposite hemisphere remains intact; compensation rapidly taking place, so that vision is again possible with either eye as before. On destruction of the angular gyrus in both hemispheres, however, the loss of vision is complete and permanent, so long at least as it is possible to maintain the animal under observation."

This opinion is not endorsed by the authors who detail Expts. XLV. to LII., which they performed in order to test the question. Their conclusions do not quite agree either with those of Ferrier or those of Munk, and they admit that their conclusions are not so satisfactory in the case of monkeys as in those of dogs. First, they have never seen blindness follow removal of the angular gyrus on one or both sides. This is totally opposed to Ferrier. Next, they think that removal of the angular gyrus (with or without a small portion of the occipital lobe—Expts. I. and LII.) is followed by hemiopia, or relative blindness of the half of the retinae corresponding with the side operated on. Munk first observed the phenomenon of hemiopia, but the authors are at issue with him to this extent, that Munk assumes that it is only destruction of the occipital lobes which can induce disturbance of

vision (amnesia of visual images, according to him), while they attribute it to destruction of the angular gyrus. They therefore conclude that the visual centre in monkeys is not confined to the occipital lobes, but embraces also the angular gyri.

As regards the auditory centre, they have no decided views to advance. They, however, think it probable that, like the visual centre, it acts bilaterally by semi-decussation of the auditory fibres, and they lay some stress on the fact that the temporo-sphenoidal lobes are anatomically correspondent in monkeys with the third external convolution in dogs, which they have determined to be the seat of the auditory faculty in these animals.

The authors recapitulate their conclusions as follows :—

1. The visual centre in dogs is represented by a long zone of the cortex of the second external convolution, which extends from about the frontal portion to the occipital lobe. That of monkeys probably comprehends not only all the angular gyrus, but also a great part, if not the whole, of the convexity of the contiguous occipital lobe.

2. The auditory centre in dogs is certainly represented by the upper and posterior part of the third external convolution; but it is not certain that it may not extend beyond these limits. In monkeys this centre is presumably contained in the region homologous with that of dogs, i.e. in a zone just external to the visual centre represented by the middle and superior temporo-sphenoidal convolutions.

3. The visual and auditory centres, both in dogs and monkeys, are electrically excitable in the whole of their extent, though the reactions obtained at different points vary not only in degree, but often even in character.

4. These reactions may possibly be due to reflex movements consequent on visual or auditory sensations; but it is much more likely that they depend on direct excitement of the special motor centres of the muscles affected, centres probably included in the visual and auditory regions.

5. Unilateral destruction of the visual zone in dogs induces at once almost complete amaurosis in the eye of the opposite side, and slight amblyopia of the eye of the same side. In

monkeys, on the other hand, unilateral destruction is followed by bilateral hemiopia of the half of the retinal field on the side operated on. It appears therefore likely that almost complete crossing obtains in the fibres of the optic nerves of dogs, and semi-decussation in those of monkeys, wherever or however the semi-decussation may occur.

6. Munk's view of psychic blindness (amnesia of visual representations) is not tenable, since the blindness produced by ablation is not only psychic, but implies also affection of visual perceptions. No change observable by the ophthalmoscope in the fundus of the injured eye.

7. Bilateral destruction of the visual zone in dogs, if very extensive, produces at once almost complete and bilateral amaurosis. In monkeys, incomplete bilateral destruction of the visual zone produces simple bilateral amblyopia.

8. Analogous effects are produced by similar treatment of the auditory centres in dogs.

9. All the effects, whether of uni- or bilateral destruction (both in dogs and monkeys) pass off in time, their duration being proportional to the intensity of the phenomena and the amount of destruction of the centre. It is not certain whether perfect compensation occurs.

10. Compensation for the effects of unilateral destruction of auditory and visual centres is effected, at least partly, by increased action of the opposite side. For, on removal of the second half of the centres, blindness and deafness occur on both sides.

11. Compensation after incomplete bilateral mutilation is effected by those parts of the centres which remain intact. Should future investigation show recovery after complete bilateral mutilation, it would be necessary to suppose that it is effected by the basal ganglia, in accordance with the authors' views regarding recovery after complete ablation of the psychomotor centres.

To settle this last question the authors performed Expt. LIII., in which they succeeded in removing both angular gyri and occipital lobes in a monkey. Vision was partly retained, and partly rapidly reacquired. From which they infer the justice of their former opinion that the basal ganglia take on

an increase of their functional value and become centres of vision.

A. RABAGLIATI.

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*The Colour Sense: its Origin and Development.* BY GRANT ALLEN, B.A. London, Trübner and Co. 1879.

THIS work is the tenth volume of the "English and Foreign Philosophical Library," and, though it differs much from its predecessors in the nature of its subject matter, being devoted to Applied, instead of Pure, Philosophy, yet the importance of the doctrine of evolution to more abstract speculation fully entitles a book like the present, which forms a highly interesting chapter in the history of the development of living beings, to a place in the series among which it has appeared.

As mere matters of fact, the contents cannot perhaps lay claim to much absolute freshness, for they are mostly drawn from well-known recent works on natural history; but there is undoubtedly much genuine originality in the way in which the facts are strung together; so that, even though the laws and modes in which evolution has occurred in the organic kingdom may be already familiar, yet they are here shown, in the department now treated of, to have an extent of application that would scarcely have been expected: the chief feature of the book being that the factors are described not so much separately, as has hitherto usually been done, but in their mutual dependence and interaction, the individual links being shown really to belong to a long and compact chain; the result of the whole being an agreeable, clearly-drawn picture of the series of events by which we obtained our principal sense.

But the greatest charm lies not solely, and perhaps not chiefly, in the leading argument itself, but in the general clearness of exposition of the details, and the artistic way in which the description is made, as well as in the abundant seasoning with keen inductive evidence, from which the author seeks to obtain the proofs of the several stages of his argument.

The author seems to aim at throwing the brilliant rays of the sun upon a region hitherto lighted only by diffused reflected beams; and, although he has occasionally to displace

another luminary, the change is one almost from darkness to brilliance.

To give some notion of the aim, scope, and method of the work, we will try to pick up the thread of the argument by the end, and unravel it continuously, step by step.

Starting from the admitted fact that many animals, besides man, not only can discriminate between varieties of colour, but also derive enjoyment from the sensation of bright hues, the author lays himself out to solve this twofold problem—why animals are sensitive to colour at all; and why certain sensations of colour should yield pleasure, while others do not? The solution is sought by first establishing empirically and inductively certain apparent facts of natural history; and then showing how the observed relations are explicable by, and fully accord with what might have been deduced from, well-known physiological principles. The problem opens thus: to find what is essential to any particular source of pleasure, it is needful to know what is pleasure in general; it is defined as “the psychical aspect of an ultimate physiological fact, . . . the unimpeded activity of a fully-nurtured and not overworked organ, in structural connection with the cerebro-spinal or other sentient central nervous system.” How, then, could such a structure, with colour perception as its function, come into existence? Organic evolution is of course taken for granted. Essential to such a process is the repeated stimulation by some object which causes benefit to the individual, for “no faculty can be originally developed for mere useless exercise in unessential acts.” We must therefore find out what objects exist in nature, giving forth bright colour, and also affording advantage to animals. On making the necessary survey we are met by the striking fact that such conditions are fulfilled by only a few substances—viz. some members of the organic world—to wit, certain flowers, fruits, and animals: for though the vault of the sky, the rainbow, and most gems present pure and brilliant tints, yet the former are too common, the latter too rare, and all too indifferent in value, to produce any impression on animals in whom a highly developed æsthetic faculty does not already exist. Even the green vegetable foliage would be too uniform and too indiffe-

rent to call for regard. At one time (e.g. in the Carboniferous period) there was probably no colour in the vegetable kingdom besides a nearly unvaried green, and probably no colour sense in animals either. How, then, could bright colours be brought into existence? Recently acquired knowledge—especially Darwin's and Lubbock's observations—have thrown much light on these processes; and the author undertakes to prove that bright colour has arisen in plants simply to attract the notice of animals; and that the sense organ to receive such impressions has developed in animals in correspondence with the increasing stimulation. The object of plants in seeking the attention of animals is to procure aid in cross-fertilisation. The object of animals in seeking plants is to procure food. At first, all plants are self-fertilised, but this produces less vigorous individuals than cross-fertilisation; if, therefore, the latter mode were to replace the first, in any species, natural selection would favour the new at the expense of the old type. The advantages of cross-fertilisation are so great that almost any way of effecting it would be attended with benefit. Two expedients have offered themselves, and been adopted in different cases, to procure the necessary dispersion of pollen—(a) the wind, (b) insects. These three modes of fertilisation correspond fairly with three palæontological periods, which may therefore be called those of—(1) self-fertilised, (2) anemophilous, (3) entomophilous plants. Of the two latter, the last is by far the best, being surer, and less wasteful: the plants in which it takes place will get an immense start over the rest in the race for life; and thus an excellent opportunity would be afforded for the operation of natural selection, if only the necessary variations could be brought about. There has probably been not much difficulty in this respect. Insects had probably been already in the habit of visiting plants, to obtain food in shape of pollen. Now, during the process of reproduction, chemical changes take place in plants, oxidation alone occurs instead of reduction, with the formation of sugar, other colouring matters in place of chlorophyll, &c. This colouring matter contrasts with the green of the surrounding leaves, and serves as a beacon to the incipient colour sense of the visiting insects, in whom the sense now goes on to improve, under the influence



of habit and natural selection. Any increase of sugar, also, will act as an extra inducement to insects to pay visits to the plants producing it, as well as being an economy in pollen to the plants; and thus an extra, otherwise useless, supply comes to be often formed, and these plants will be indicated to insects by peculiar or extra bright colour. But colour is not the only means of attracting animals that is resorted to. The incipient sense of smell is also appealed to, and the perfume and corresponding organ of smell proceed to develop by gradual variation and natural selection, just as colour and its sense organ have been shown to do. So great does the author believe to be the resources possessed by plants, that he states in general terms that any part of a plant that will be benefited by the notice of animals, will forthwith tend to develop in it bright colour and sweet smell. Though the bright colours are at first produced in all parts of the plant indiscriminately, they gradually become chiefly concentrated in certain parts—usually a few leaves, which become the petals. The primitive flowers were regular and polypetalous; but became irregular and gamopetalous in becoming more adapted to the insects visiting them, in order that the end aimed at shall be more readily brought about. In this way, the author believes the senses of sight for colour, smell, and taste to have arisen. The same device has arisen separately in monocotyledons and dicotyledons, and has probably originated afresh innumerable times.

Something similar has taken place in the case of fruits. The period of maturation of seed is the second critical epoch in the life of a plant, in which its fortune depends much on circumstances. As a large proportion of all mature seeds perish from lack of nourishment for the embryo, any means of ensuring a supply for the new plant would be attended with advantage to the species. It has been more or less completely effected or replaced in three chief ways: 1. Replacing the seed by an underground stem with a supply of nutriment and tendency to germinate. 2. Supplying a store of nutriment to the germ for its use when activity commences. 3. Distribution of the ripe seeds over a wider area, so that the chances of getting sufficient nutriment in the soil would be increased. But, in the first and second cases the extra supply of food

tempts the appetite of animals; for the store is equally good food for both, and is a richer and more economical kind than animals could otherwise obtain; this method would, after all, often be injurious to the vegetable species, unless some means of protection against these depredations be acquired. This in many cases has been done—in the former mode by the store remaining underground, out of sight; in the latter, the seeds having acquired a hard coat, to defy the teeth, a dull colour to escape the notice, and often a bitter outer husk to displease the taste of the animal who would eat them: this is the mode of the genesis of the nut. But in the third case, dispersion may be effected either by the wind—in accordance with which many changes for facility in conveyance have come to pass in seeds; or by the aid of animals—drawn to the seed in search of food; but, though animals will pay no visits unless they are rewarded by food, yet no advantage will accrue to the plant if the embryo itself be destroyed. It is therefore essential to allure and attract animals, and yet protect the germ. The accomplishment of these ends results in the formation of a fruit (using the term in its popular sense). The actual process is much like that of the origin of flowers—out of the same tendencies to variation as were previously described, and which occur at this period also, result the formation of sugar, bright colouring matter, and perfume, to appeal to the senses of taste, sight, and smell of animals, in this case, mostly birds and mammals. But in them, fruits do not work so complete a change as flowers do in insects; for the vertebrata have already probably some sense for colour, inherited from some common marine invertebrate ancestor, in whom the colour sense had probably been gradually awakened in a precisely similar manner, by the attraction of bright coloured food. The colour sense being thus produced, pleasure results from its exercise, according to the law enunciated above. The author is careful to make this statement, to prevent his being understood to mean that the pleasure of bright colour was merely extrinsic—due to the transference of the pleasure of gratified appetite and successful search, produced by the finding and eating of fruit, to the visual sensations by which such success has been attended, according to the law of association.

When the mere exercise of the sense has thus become pleasurable, it is exerted in other ways: notably in sexual selection; by which process, the continual preferment of bright-coloured mates by the females has led to the establishment of bright-hued species; as in plants, an original tendency to produce colouring-matter existed—especially in the tegument: many waste matters of the organism being pigmented and attracted to and fixed in the skin as a vicarious excretory organ.

But colour, except in a few cases, does not attain a higher degree of intrinsic beauty in animals below man; in him, however, the love of colour for itself leads to the decoration of his person, house and property with bright colour; and lastly, to the appreciation of beauty in the gorgeous hues of the sky at sunset.

Such is a brief and imperfect outline of Mr. Allen's theory; the chief points on which it hangs are the soundness of his physiological theory of pleasure—which seems to be a fair statement of the truth, although he perhaps does scarcely sufficient justice to its rival—the association theory of pleasure; for there is plenty of room for both. But its main support, to which the chief part of the book is devoted, is the accuracy of correspondence between possession of colour sense and habits of eating coloured objects. It is in discussion of these matters that the author displays his best reasonings and skill for investigation—shown by devising passive experiments; and he gives many examples of the different inductive methods. Although he certainly traces a most accurate correlation in numerous crucial cases, yet he acknowledges the occasional apparent impossibility of explanation of others, and even rarely a seeming opposition. However, it is very likely that an extensive acquaintance with natural history might clear away these obstacles; and at any rate this would be the best way of obtaining a decision.

The positive part of his theory being established, Mr. Allen proceeds—and with success—to combat the rival scheme of Gladstone, Magnus, and Geiger: who hold that the linguistic evidence of the Vedas and Zend Avesta, the Bible and Homer, shows that in early historical times man had little or no

appreciation for differences in colour; but that, since then, he has gradually acquired a perception for red, yellow, green, in order, and lastly blue.

But by reference to numerous existing savage races, as well as to still earlier historical and even prehistoric peoples, he shows almost with certainty that they really had a well-developed taste for variety of colours; though their speech affords insufficient evidence.

The final chapter contains a description of the mode of growth of the colour vocabulary; but is not much more than a particular example of the process of word-growth already described by Adam Smith and his successors.

Though probably all will admit the author's explanation of the important bearing of coloured food in developing the colour sense, yet all his readers may not feel satisfied that it has been the sole mode. For, though it might account for the greater degree of pleasure derivable from red, yellow, and blue, than from green, yet, according to his physiological data, we should have expected that the pleasure derivable from a sense would be proportional to its degree of development, and therefore to its keenness of discrimination, i. e. of discerning fine relations and minute differences; yet this does not appear to be greater for bright colours than for others. Also, it would be very difficult to explain, by this theory alone, the converse facts of distaste. Also it would still leave some other senses—especially that of hearing—still unexplained,—and almost hopelessly so. But in the latter case a large proportion of the sensation of harmony seems to result from physical harmony of sounds; before dismissing altogether, therefore, the opinion that a similar condition—of harmony—might be the physiological conditions of pleasurable colour, it would perhaps be advisable to wait until the physical basis of vision has become better known to us; and perhaps nothing would lead more to this than a thorough comparison of the physiology of our two chief senses.

F. L. BENHAM.

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## Clinical Cases.

### A CASE OF DOUBLE OPTIC NEURITIS, WITHOUT GROSS CEREBRAL LESION, WITH REMARKS UPON THE IMMEDIATE CAUSATION OF OPTIC NEURITIS. (*Illustrated.*)

BY STEPHEN MACKENZIE, M.D.,

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THE following case is of such interest that I hope I shall be pardoned for the detail with which I have narrated it. The patient had evidently been for a long time on the border-land of insanity, and the acute maniacal delirium which terminated the case may be taken to indicate that she had passed beyond the border. As in so many cases in which the mental faculties are at fault, no coarse anatomical changes were found in the brain beyond wasting of the organ. Microscopic examination, however, showed changes which fully accounted for the symptoms presented. The long period over which the mental symptoms extended would point to the brain being primarily at fault. Atrophy of the brain such as she had is sometimes found in arterio-capillary fibrosis. No such condition was, however, present in her case. Her body was well nourished, the kidneys healthy, the left ventricle not hypertrophied. In cases of atrophied brain and cord in connection with arterio-capillary fibrosis, similar exudative changes have been found by Sir William Gull and Dr. Sutton, and it is noteworthy that the end of such cases is frequently ushered in with acute symptoms referable to the brain and spinal cord. Indeed this seems to be in accordance with a broad pathological law that tissues in a state of decay or of impaired nutritive activity are prone to become inflamed.

The symptoms presented at one time resembled hysteria, and the attacks were thought by some to be hysterical. In this respect the case recalls the valuable one recorded by Dr. A. Hughes Bennett, in the first number of 'BRAIN.' In his case there was a tumour; in the present there were marked,

though minute, structural changes in the brain. In many of the attacks the patient had spasm of the pharyngeal and laryngeal muscles, reminding one of hydrophobia. The nurses of the ward, who have seen three or four cases of hydrophobia in the hospital, were struck by the resemblance. The case is as follows:

Marie Schwabe, a Frenchwoman, a native of Paris, aged 27. When I saw her, in September 1878, she was a woman of somewhat short stature, inclined to be stout; dark hair, almost black, and with long hairs growing from upper lip and chin; teeth good. Her expression was that of a woman who had suffered a great deal—an expression of anxiety and despair. Patient's father and mother living; the former had been afflicted with hemiplegia for some years. She has two sisters and one brother, living and healthy; no history of phthisis, epilepsy, or insanity in family. Her previous history was obtained chiefly from her husband, an intelligent clerk.

She had good health and manifested no mental peculiarity, as far as is known, up to the time of her marriage, at 18. During the whole of her married life, however, she used constantly to express to her husband a dread that she would go mad; and "she used to dream almost every night of mountains, serpents, lions, and other animals, with the heads of people she knew." She used also to complain of a sense of suffocation if she was in a room with the door shut. Her memory was impaired for ordinary affairs, but was unnaturally acute in recalling anything bad of those she knew. Was not pregnant until three years after her marriage. When five months advanced in pregnancy she had a very bad miscarriage, without assignable cause. She had been complaining of obscure pains over the region of the uterus for some fourteen days previously. She was attended by a doctor, but lost a good deal of blood. The following day, on her husband coming home in the afternoon, she complained that the nurse whom he had engaged to look after her had been drinking brandy, and was drunk. (This was quite imaginary.) She then, in a wild manner, said several times, "You give her coffee." She became extremely excited, sprang up in bed, tore down her clothes, which were hanging on a nail by her bedside, and said, "I want to go away—I want to go away." She then had a kind of fit, similar to those she had subsequently in the hospital—repeatedly opening and shutting her mouth, stoppage of respiration, turning pale, violent struggling, and "became insensible." The attack lasted about three or four minutes: she did not bite her tongue. She gradually recovered her senses, and remembered nothing of what had occurred. A few hours after this she had a laughing fit, and repeatedly said to her husband, "You

laugh with me!" Next day she had "convulsions" from time to time, and a peculiar twitching of the muscles of her face. This twitching lasted continuously for one week, and since that time to her death she had this twitching, if excited. She improved a good deal for three weeks after this, when she again became nervous; and one day, on her husband opening the door, said she believed that the members of the club to which he belonged would poison her, and expressed a great dread of everybody she knew.

Menstruation had been irregular since her miscarriage; she had excessive flow every two or three weeks. After her miscarriage she walked with difficulty, and vomited after her food. She consulted a doctor, who placed a pessary in position, with temporary relief. She went into the country for a few weeks, but at the end of that time became nervous again, "lost the use of her limbs," and suffered constantly from severe headache. She says sometimes the pain was so severe she could not think; she used to say stupid things which she afterwards remembered. About nine months before her death she began to take a great dislike to people whom she saw for the first time, avoided persons her husband brought home to supper, and complained to him that he forced their society upon her. She took to eating the tops of lucifer matches (a favourite continental method of committing suicide), and her husband had to remove them forcibly from her. The least noise, or any one speaking to her, made her very nervous. She says that now she is not able to control her feelings, and burst out crying if refused anything she asks for, and this distresses her a good deal. For three months before admission took narcotics at bedtime, but even then has not had proper sleep, always being restless, and having disturbing dreams, seeing strange figures, &c., which have caused her to wake suddenly, and become very agitated. In one of her dreams she thought she was eating her own brain. For three days previous to admission she had suffered so much from headache that she could not be left alone, and she vomited after everything she took. The headache had been far greater than previously the six weeks preceding admission. Throughout her illness she was troubled a good deal by constipation.

The patient had had an operation performed on her right eye (for strabismus?) in childhood. For six months preceding admission she had been complaining of weakness of her eyes, especially the right, but ever since her husband has known her, she has had to put her hand over her right eye on reading or writing. There is no evidence to show that her left eye had been failing previous to admission, beyond the fact that on the day of admission she had been previously to see some

friends, and on entering the room had complained that she could not see any one.

A week after her miscarriage a Hodge's pessary was introduced, and she wore it for some months until it slipped out during defæcation. She then had it replaced, and wore it until the end of last March, when she was told the womb was in place. A few weeks later, when straining at stool, she felt the womb fall. The pessary was again introduced, and she wore it until a few days before admission, when she took it out herself.

She applied at the obstetric department of the London Hospital, on account of her uterine symptoms and of increasing pain in the head, and was admitted under the care of Dr. Herman, August 28th, 1878.

She then complained of great pain in left ovarian region, and down left leg and side, pain in the head, and dizziness. On vaginal examination, the uterus was found to be retroflexed, movable, and the fundus large and exceedingly tender. There was an erosion on each lip of the cervix, with a copious glairy discharge from cervical canal; the cervix was blue and congested. The cervix was scarified two or three times, and a mixture prescribed consisting of sulphate of magnesia and bromide of potassium. The uterine congestion having subsided, a Hodge's pessary was fixed, and the womb found to be well-retained in position.

Her other symptoms, however, increased; the headache was very severe, and she frequently vomited—sometimes clear fluid, sometimes green fluid. I was therefore asked to see her on Sept. 22nd.

I found her exceedingly nervous, starting whenever any one approached her, and looking extremely frightened. She complained very much of her head, and implored relief. Physical examination of her chest and abdomen revealed no important changes. Her tongue was coated with dark brown fur; bowels much constipated. There was no œdema of feet.

Ophthalmoscopic examination of left eye showed swelling of the optic nerve, the margin of the disc being wholly lost to sight. Owing to patient's restlessness, no detailed examination could be made. The right eye could not be examined with the ophthalmoscope, owing to old iritic adhesions.

Her vision was not tested by types, but the following facts indicate its condition. On her admission (about a month before I saw her, and before the neuritis was discovered) she wrote to her husband: this letter was a long one, and written in a small ordinary hand. Her letters gradually became shorter, and the type larger and larger. In three weeks she could not see to write at all, and got the patient in the next



bed to write for her; from time to time she asked her husband to write to her in larger type, and complained even then. She had to hold his letters quite close to her eyes. She could see quite well previous to admission: she could use her right eye for near objects (to read), but not for distant objects.

Her temperature since the beginning of September has averaged nearly 100° Fahr., the maximum being 101°, the minimum 98·4°: the evening temperature being usually higher than the morning. Urine, s. g. 1026. Urea 3·3 per cent. A very faint trace of albumen, a little epithelium only in deposit.

She was transferred to my care.

*Sept. 30th, 12.30.*—She screamed out, was purple in the face, and tried to tear her pocket-handkerchief. When seen by the medical officer, she was quite quiet, but not unconscious. When spoken to, answered questions quite rationally. She asked to have her husband sent for. At times she cried out; at others made a peculiar yelping noise. When I came to her bedside in the afternoon, she was lying in bed quite quietly, with a very anxious expression of countenance. She said she felt comfortable, but was very anxious that something should be done to relieve her head. Whilst talking to her she was seized with spasm of the laryngeal and pharyngeal muscles; her mouth would be suddenly opened two or three times in succession; she made a gasping noise, and occasionally a sudden inspiration, accompanied by spasm of the larynx, which produced a yelping or barking noise. The spasms appeared occasionally like exaggerated hiccough. Some of the noises she made occurred during expiration. She said "Give me some water," and when a glass of water was placed in her hands, she could drink, swallowing without any great difficulty, but her agitation and tremor made it difficult for her to hold the glass to her lips. She seemed very anxious about herself, and said inquiringly, "I am not dying?" She frequently asked for water, and each time drank some. The spasms continued for about a quarter of an hour, as long as these notes were being made. She complained of a burning sensation, "like a fire," in her chest. She occasionally put her handkerchief into her mouth and bit it.

The following mixture was ordered to be taken every 3 hours:

℞ Potassii Bromidi  
Chloralis Hydratis ā gr. x.  
Acid. Hydrocyanic. dil. ℥. iij.  
Aquam ad ʒj.

8.30 P.M.—She complains during intervals of quiet that the bowels have not moved; she still has burning pain over

epigastrium, and severe headache. Her teeth are chattering (temp.  $99.7^{\circ}$ ). She keeps twisting her pocket-handkerchief between her fingers. She has had a little quiet sleep since taking her medicine, but on waking up the spasm of the pharyngeal and laryngeal muscles returned. She can talk quite sensibly during the intervals. On giving her some water she drank it readily, but spasm followed immediately. She caught at the bedclothes, and made a peculiar rasping or barking noise, and then threw herself back in bed. She says the cause of her attack was seeing a big woman with a shawl over her head, walking up and down in front of her bed last evening, and that the woman struck her head with something black. She has not vomited to-day. Pulse 96, small.

2 A.M.—House Physician called to her bedside. He found her yelping, and on giving her something to drink, spasm immediately followed. After an interval of quiet, she began laughing, and continued to do so for about five minutes. She said, "You don't laugh; you see I do!" This was followed by a fit of crying.

*Oct. 1st.*—She has had about four hours of quiet sleep in all, but each time on waking up immediately began to make a yelping and barking noise. During intervals of quiet she protrudes her tongue when asked to do so. She says the pain in her head is still very bad, and she adds, "but I have more reason this morning." She complains of something which she says is grasping her throat with two hands, and also of severe burning pain over epigastrium.

9 P.M.—After  $\frac{1}{4}$  grain of morphia hypodermically, patient was sleeping quietly, and afterwards passed a good night.

*Oct. 2nd.*—On coming to her bed this morning she was quiet, but immediately after being spoken to began yelping. This continued for about five minutes, and was followed by a fit of laughing, during which she threw herself about in bed. She appears afraid lest she should be going to die, and continually asks if she shall get better. She has not vomited since yesterday; bowels were open last night after an enema. Passes her urine freely. The mixture was stopped until this morning. She still complains of severe headache. Is able to drink without any spasm being produced, but a short time afterwards made the same noises as previously.

*Oct. 3rd.*—Slept until one o'clock this morning, when on waking, began laughing and crying. On her husband persuading her to try and sleep, she became composed, and passed a good night. Has had no more vomiting. Drank a pint of milk, with only a little spasm. A hypodermic injection of morphia, gr.  $\frac{1}{4}$ , was given at 11 A.M. This morning, on coming

to her bedside, she immediately had a fit of laughing, during which she beat the bed with both hands. This was followed by shrieking out at the top of her voice, and then she said, "Boo, boo." Still complains of headache.

*Oct. 4th.*—Passed a quiet night; has not vomited, and says headache is better. She was lying quite quiet, when, on another patient in the ward laughing, she immediately broke out laughing, screaming at the top of her voice, beating the bed with both hands. Has taken some milk. Passed her urine this morning.

When I saw her in the afternoon, I dictated, "patient was always pale, but now her face is white; there are dark halos under her orbits. The left eyelids are swollen and red. Altogether she looks extremely haggard, and as though she had had but little sleep for some time. She is still very much excited, tosses herself about in bed, and frequently strikes the bed with both hands a number of times in succession, and with great force. Her tongue is coated; there is slight sordes on her lips. The bowels have not acted for three or four days. Left pupil contracted, and an ophthalmoscopic examination impracticable."

*Oct. 5th.*—Had an enema last night, after which her bowels acted twice. Had a quiet night until 4 A.M. The pessary she was wearing was removed last night, since then she says she has been easier. On coming to her bed this morning she immediately began to shriek out, to beat the bed with her hands, and then to throw herself back in bed. It is very difficult to keep her in bed, and she struggles violently when any attempts are made to restrain her. Has passed water. She keeps crying out about "the naughty woman," and says "she was very naughty to make me so ill." This was followed by a fit of laughing. Has not vomited.

Evening: was quiet on coming to her bedside, but immediately began laughing immoderately.

*Oct. 6th.*—Passed a quiet night after an injection of morphia, but is in much the same condition this morning; first laughing, and then crying. She requests every one who comes near her to laugh, and then does so herself; she afterwards bursts out screaming as loud as she can, becomes very violent, and at times it is a difficult matter to keep her in bed. No vomiting. It became necessary to place her in a separate ward, as her incessant noise disturbed the other patients. She passed a large quantity of urine yesterday afternoon; has taken a fair amount of nourishment.

*Oct. 7th.*—Slept quietly until 2 A.M., when she became very noisy, laughing and crying. She is very irritable. Passed her urine twice yesterday, and her bowels have been freely opened.

*Oct. 8th.*—Passed a quiet night: no pain in head this morning. Has passed her urine; bowels open in the night. She has been much quieter this morning, but called her husband “a fish.” She has not screamed so much this morning. Says she is losing her reason. No vomiting; takes nourishment very well.

*Oct. 9th.*—Passed a quiet night. Is more tranquil this morning. Has a haggard, worn look. Has not vomited. Has taken a basin of arrowroot this morning. Both upper eyelids are now swollen. Drinks without any spasm being produced. Tongue coated. Passes urine very freely, but in varying positions.

*Oct. 11th.*—Passed a quiet night. No albumen in urine: no vomiting. Says she has no pain in the head. Tongue thickly coated. Passes urine freely. Her expression is still more haggard.

*Oct. 14th.*—Passed a very restless night, in spite of morphia and bromide of potassium. Bowels confined: passes urine freely. Eyelids less œdematous. Complains less of pain in head. Says she is better. She does not scream this morning, but makes a groaning noise. Will not protrude her tongue when asked to do so. Takes nourishment.

*Oct. 16th.*—Passed a restless night. Her husband (who is almost constantly with her) says she is able to swallow, but after having done so makes a gurgling noise, and then anything she has taken returns. She has a worn-out look. Her pulse is small, 96. She does not appear to have strength to scream. Has passed her urine and motion in the bed. No paralysis of limbs. Does not complain of pain in the head, but still complains of pain over epigastrium. Recognises her husband and the nurse.

*Oct. 17th.*—Passed a quiet night. Has taken a fair amount of nourishment. When asked if she is better, says “Yes.” She looks exceedingly ill; her pulse is small; feet and hands cold. She is very restless. Passes both urine and fæces in bed. Has been screaming more this morning.

*Oct. 18th.*—Passed a restless night. Has been screaming all night. Bowels moved freely yesterday. Has taken a fair amount of nourishment.

*Oct. 19th.*—Passed a quiet night, except at intervals. She is restless in bed this morning. On being asked if she has any pain, she does not speak, but shakes her head. Passes her urine in the bed. Takes nourishment.

*Oct. 21st.*—The patient passed a fairly quiet night. Nurse reports that several times, without any cause, her breathing stopped, and she turned purple in the face. After a few seconds there was a gurgling in the throat, and breathing

recommenced by her taking a long inspiration. This was observed several times (Cheyne-Stokes respiration). She does not answer when spoken to. Passes her urine in bed: bowels not open for several days. No paralysis of any of her limbs. Pulse 84, small and feeble. She cannot be roused. Swallows with difficulty. Feet and hands and surface of body cold. She is getting rapidly worse.

2 P.M.—Patient evidently sinking.

Died early in evening.

Her temperature averaged about 100° Fahr., the maximum being 101·6°, from Sept. 22 to Oct. 6. After this date she was too restless to admit of it being taken. On the two occasions her urine was examined, its specific gravity was 1026 and 1025 respectively, and contained 3·3 and 3·6 per cent. of urea. The quantity of urine secreted could not be measured. On the first occasion a very faint trace of albumen was present, on the second none.

The treatment consisted of large doses of potassium bromide, with and without chloral, of subcutaneous injections of morphia, of aperients as required, and the frequent administration of easily digested food.

The *necropsy* was made on the 22nd, at 3.30 P.M., by Dr. Sutton, in my presence. The following is Dr. Sutton's report, extracted from the Post-mortem Book:—

“Atrophied brain. Fatty deposition on right ventricle of heart. Much subcutaneous fat.

“Largely developed body, with large regular features. Limbs seemingly somewhat above the average bulk. No œdema. The subcutaneous fat over thorax and abdomen was about one inch thick.

“Skull and dura mater normal. Pia mater milky-looking and semi-opaque. The brain convolutions were much atrophied over anterior two-thirds of the hemispheres. The sulci were much widened. The grey matter of brain on section looks normal, merely venously congested, and the brain substance on section looked normal throughout. The vessels at the base were seemingly normal.

“The heart weighed 8½ oz.: it looked smaller than normal. There was much fat on the right ventricle near the apex: it had grown into the wall of the right ventricle, leaving scarcely any visible muscle: muscle more natural above. The left side of heart was natural, excepting that the wall looked thin.

“Lungs œdematous, and hypostatic pneumonia at back part of one.

“Liver and spleen normal. Kidneys normal. The mucous membrane of stomach was studded with a number of yellowish

masses, the size of pins' heads; they looked like stomach tubes distended with fat."

Dr. Sutton appends the following note to the case:—

"There was no evidence to show that the atrophy in this case was part of a general atrophic process. The post-mortem examination would leave it uncertain as to whether the fatty accumulation and degeneration occurred antecedently, rendering the right ventricle unable to empty itself properly—leading to venous distension of the pia mater and venules of the convolutions, and by pressure of these distended veins, or otherwise, producing the atrophy of convolutions, more or less dementia, and the subsequent obesity."

*Microscopic Examination of optic nerves and retina, and of brain.*—The right optic nerve was removed for the purpose of examination. This eye had not been seen during life owing to iritic adhesions. It shows increase of nuclei of trabeculæ and nerve fibres, and considerable collection of lymphoid cells in trabeculæ. This cell infiltration extends throughout the whole length of nerve examined (a considerable length), but the cells are most aggregated below the lamina cribrosa. There is very considerable infiltration of the outer part of the internal sheath of optic nerve with round cells (lymphoid cells or leucocytes), and the fenestrated membrane has great increase of nuclei owing to this germination. Nerve fibres of optic disc swollen, and lower layers infiltrated with cells continuous with those in trabeculæ of optic nerve. Slight infiltration of ganglionic layer with lymphoid cells. Both granule layers disturbed in regularity and arrangement. Retinal blood-vessels distended with blood corpuscles. Slight infiltration of choroid at optic entrance, at the part immediately contiguous to the cell infiltration of sheath. Arteria centralis has its adventitia very slightly thickened.

Pia mater slightly infiltrated, showing some chronic change.

Brain cortex infiltrated with lymphoid cells (leucocytes), especially in second layer (small pyramidal cell layers). The lymphoid cells can be seen following the tracks of the vessels, and have obviously exuded from them: they are especially in connection with venules, slightly with capillaries. Pyramidal cells so altered as to retain no longer the shape from which they derive their name: they are represented by a collection of three, four, or more small nuclei, and some ill-defined protoplasm. In the first and second layers of cortex are some ill-defined spherical masses, staining purple with hæmatoxylin: they are apparently masses of exudation (plasma or lymph). Nuclei of blood-vessels in places germinating. Several parts of the cerebral cortex were examined and the same changes



Stephen Mackenzie delt:

Hartnack. Obj. 5, Oc. III.

Section through deeper part of Frontal Convolution showing longitudinal sections of small vessels and branches, filled with coloured and colourless corpuscles, with emigrant colourless corpuscles (leucocytes) collected around the vessels. The exuded corpuscles are represented dark as stained by logwood.





found at each. Similar changes in corpus striatum. Throughout brain-blood-vessels much gorged.

REMARKS.—The existence of double optic neuritis without coarse local cerebral disease is a point not only of interest but of considerable clinical importance. Optic neuritis is so commonly symptomatic of some adventitious intracranial product, or of some gross intracranial lesion, that a diagnosis often turns on this one point. That it is not always indicative of coarse intracranial disease cases reported by other observers show. Dr. Hughlings-Jackson, in particular, has drawn attention to this point. In reporting “A Case of Double Optic Neuritis without Cerebral Tumour,”<sup>1</sup> he quotes from a Lecture on Optic Neuritis, delivered in 1871.<sup>2</sup>

“I have been wrong several times in the diagnosis of an adventitious product within the skull, in cases where there had been found double optic neuritis; *but I have far often<sup>r</sup> been wrong by neglecting the inferences above stated to be deducible from the presence, or absence, of optic neuritis*—wrong in saying there *was* an adventitious product when the discs were normal, and wrong in saying there *was not* when there was double optic neuritis. I feel, therefore, justified in saying that double optic neuritis does point *very strongly indeed* to coarse disease inside the head [*italics in original*].

In the case I have narrated I had the benefit of Dr. Hughlings-Jackson's opinion. He kindly took charge of the patient during a short absence, and wrote to tell me on my return that he did not expect “local coarse disease” would be found in the brain after death.

But why should this patient get optic neuritis, the commonly ascribed causes being absent? This raises the question, Why does optic neuritis occur in connection with its usually acknowledged causes—tumours, abscess, &c.?

I think that in spite of the eminence and ability of its advocates, from V. Graefe downwards, the pressure theory of swelling of the optic nerves, whether called choked disc or neuritis, cannot be maintained. We see large encephalic tumours, sometimes of rapid growth, without any neuritis, and we see small tumours in distant parts of the cranial cavity—as in the cerebellum—with power to provoke the neuritis. Neither is there any ante- or post-mortem evidence, other than that of the optic nerves themselves, to bear out the theory of a Stauung's papilla. The inflammation of the optic nerves, which we see with the mirror during life and with the microscope after death, appears to me to be excited in all cases by either an extension process from the membranes of the brain

<sup>1</sup> The ‘Royal Ophth. Hosp. Rep.’ Vol. VIII., part iii. p. 445.

<sup>2</sup> ‘Med. Times and Gaz.,’ Sept. 16, 1871, p. 341.

to the optic sheath, and thence to the nerve and retina, from the brain to the optic tract or nerves directly, or by an independent affection of brain and optic nerve by a common cause acting by selection upon histologically allied structures. Of course the inflammatory changes in the optic nerves and discs are modified by the anatomical peculiarities of the part. Vascular dilatation, and serous and corpuscular exudation, though constituent factors in the process of inflammation, do not necessarily advance *pari passu*. In some cases the dilatation of vessels predominates (influenced no doubt by the condition of the vascular system of the patient at the time of the disease inducing the neuritis), whilst in others exudative changes are in excess of vascular dilatation. Hence arise differences in the appearances of the optic nerves, to which many observers attach importance. It is acknowledged, however, by most that these differences have no practical significance as indicating the nature, the position, or the duration of the intracranial lesion on which they depend, and we may therefore conveniently speak of the swelling and exudative changes of the optic nerves under the title of neuritis. To repeat, the neuritis associated with intra-cranial disease is, in my opinion, an inflammation communicated from the membranes, or from the brain itself to the optic nerves, except in the rare cases where they are independent, but due to a common cause. In some cases the implication of the membranes, especially basal, affords a ready explanation of the occurrence of the neuritis. Where the meningitis (tubercular, zymotic, traumatic, or an extension process from abscess, &c.) appears the sole encephalic lesion, this explanation may suffice without bringing in another; but at the same time it must be remembered the *symptoms* of meningitis are due to the changes wrought in the brain, and which are never absent; and so it must be borne in mind that a possibility exists that the neuritis accompanying meningitis may really be an extension process from the implicated brain. Excluding cases of primary meningitis, a secondary meningeal inflammation cannot be brought in to explain the occurrence of neuritis from tumours, as the evidence of such meningitis is frequently wholly wanting. We are therefore thrown back upon some change communicated from the brain tissue to the optic nerves, to explain the neuritis due to coarse cerebral disease, and my impression is that the inflammatory changes found in the optic nerves are in all cases (excepting cases of meningitis) either an extension process from the brain, that is to say, "a descending neuritis," or a common process shared in by the brain and optic nerves. I say my *impression*, because I am not prepared to speak dogmatically on the point without further evidence. I am engaged just now in investi-

gating this subject from the histological stand-point. I may, meanwhile, draw attention to some facts which appear to favour this view. In the first place a tumour in the brain as such cannot give rise to symptoms, except by exciting changes in the nervous tissues surrounding it. Where we have permanent paralysis, there must be destruction of nerve tissue; where there is spasm, pain, vomiting, &c., there must be disturbed nutritive activity associated with tissue changes. Whether the tissue changes around tumours are or are not to be called inflammatory is a question of terminology. There are found vascular dilatation, corpuscular infiltration, thickening of neuroglia, &c., the changes which we should regard as interstitial inflammation in any other organ than the brain. So I take it the changes found around tumours, &c., in the brain are of an inflammatory character, tending to spread in all directions or in certain directions, and in so doing frequently implicate the optic nerves, which really are modified portions of the brain outside the general cranial cavity. Of course I must not be understood to deny that a large and rapidly growing tumour will produce pressure symptoms. My object is to show that pressure cannot be the usual explanation of neuritis from local coarse intra-cranial disease.

The direction the cerebritis takes and the rate of its progression are matters of great moment which require further investigation. Whilst it would at first sight seem probable that inflammatory changes would spread in all directions around the nucleus formed by a tumour, yet it is more probable that the cerebritis spreads most in certain directions—directions of least resistance, of more direct anatomical continuity, &c. We see this in rapid changes as hæmorrhage, and in slow changes as sclerosis. A strong point in favour of the cerebritis which I assume excites optic neuritis, having a tendency to travel in special directions, is the fact that in two cases of uniocular neuritis from cerebral tumour recorded by Dr. Hughlings-Jackson,<sup>1</sup> the neuritis was on the side opposite to the tumour. On any pressure or vaso-motor theory, the neuritis should have been on the same side as the tumour, and the same may be said of the neuritis being set up by intermediation of meningitis. But the inflammation travelling down the optic tracts would explain the occurrence. Such cases of uniocular neuritis are rare, and it would be very valuable if those who have had the opportunity of seeing any completed by a post-mortem examination would place them on record.

If the cerebritis spread in various directions, we should expect the implication of other cranial nerves. It is the expe-

<sup>1</sup> 'A Physician's Notes on Ophthalmology,' Dr. J. Hughlings-Jackson, 'Reprints from Ophth. Hosp. Reports,' &c.

rience of most observers that, whereas amaurosis is common from coarse cerebral disease, deafness is rare, whilst defects of smell and taste, being much less important, are passed over with little notice. As regards nervous deafness, Dr. Hughlings-Jackson has especially remarked upon its scarcely ever occurring in connection with coarse cerebral disease. But it must be remembered that he has (as he says, he fears, in season and out of season) repeatedly called attention to the existence of double optic neuritis without defect of sight; and so it may be that, less open to our examination, inflammatory changes occur in the auditory and other cranial nerves without being detected. It is significant that of the only two cases Dr. Hughlings-Jackson has recorded (so far as I know), in one case he expressly states that he could not account for there being deafness of both sides, seeing there was only disease (tumour) on one side; in the other case he only states (in reprint) that the patient was blind and deaf, and that at the autopsy a tumour was found in one sphenoidal fossa, reaching as far back as the seventh pair of nerves, producing changes in the hemisphere which had reached the auditory nerve. The other cranial nerves, as the third, fourth, sixth, &c., are sometimes paralysed without any explanation of the causation, such as meningitis, &c. It may be in such cases the inflammatory process has reached them from the brain, setting up neuritis.

We know little concerning the circumstances which influence the rate at which the cerebritis spreads from the vicinity of tumours. The difficulties underlying this point are very great. We only know of the existence of intracranial tumours at all from the changes excited in the brain-substance around them. We know that optic neuritis may be one of the earliest signs of a tumour, and at the same time we know a tumour may exist for a long time without exciting neuritis. I have watched a patient of Dr. Hughlings-Jackson's, in whom a cerebral tumour was subsequently found, who had symptoms (unilateral convulsions beginning in one great toe) indicating its existence and precise locality several years before neuritis occurred. Nearness to the optic tracts is probably one circumstance which influences the period of occurrence of optic neuritis. Cases of cerebral tumours without neuritis are probably cases in which the cerebritis has not reached the optic nerves.

The present state of our knowledge, then, I hold is not inconsistent with the neuritis occurring in connection with coarse cerebral disease being due to an extension of the inflammation to the optic nerves from the inflamed brain around the encephalic lesion causing the neuritis by direct continuity of tissue, though I am aware how deficient is the

direct evidence of such causation. What is wanted is careful microscopical examination of continuity of cerebral tissue from the tumour to the optic discs in many cases with and without neuritis. I am collecting material for such examination. I would, however, refer the reader to Dr. Clifford Allbutt's work, 'The Ophthalmoscope in Diseases of the Nervous System,' where the subject is discussed with such completeness and ability. Dr. Allbutt, however, draws a wide distinction, both as regards the appearances and causation, between the choked disc and descending neuritis, and believes the latter only to occur by propagation from the membranes or cerebral tissue.

But how are we to explain the cases where optic neuritis occurs without its usual exciting causes? I think the microscopical examination of the brain in my case affords some solution. It cannot have failed to have attracted attention of observers that, apart from neuritis, the changes which occur in the optic nerve and retina are alike in kind with those co-existing in the brain. We see this in Bright's disease, in diabetes, in leuchæmia, in idiopathic anæmia, in ague, in purpura, &c.

In the above case the changes I have described and depicted in the brain are of a kind which in any other organ would be regarded as inflammatory. Fashion is largely responsible for our terminology. Similar exudative changes in the liver or kidney, in the skin, the stomach, or the optic nerves, would undoubtedly receive the name of inflammatory, and we should speak of interstitial hepatitis or nephritis, or of dermatitis, interstitial gastritis, optic neuritis. So, I take it, the changes found in the brain in the above case show a condition of cerebritis or encephalitis. Though to the naked eye there appeared only atrophy of the brain, the microscope showed a corpuscular infiltration of an inflammatory character. But it may be objected, How could the change be inflammatory when the brain was atrophied, seeing that inflammation causes swelling (tumour)? I need only instance interstitial nephritis and hepatitis as examples in which inflammation and shrinking go hand in hand together. The changes need not be exactly contemporaneous throughout the brain; whilst exudation is taking place at some parts, other parts previously affected may be undergoing contraction. The microscopical changes in the above case in the brain and optic nerve are precisely of the same character. We saw with the ophthalmoscope in the eye during life what we could only see in the brain with the microscope after death. As to whether the changes in the optic nerves were sequential to, or contemporaneous with, the changes in the brain, there is no precise evidence to show, though the longer duration of

cerebral symptoms and the shrinking of the brain would incline me to believe that the optic neuritis was secondary—an extension-process from the brain. In Dr. Hughlings-Jackson's patient, to which I have referred, there was no wasting of brain or gross-naked eye change. Dr. Sutton examined the brain microscopically, and reported that sections of the convolutions of anterior lobe showed a large number of nuclei, about the size of white blood-cells, some appeared to be dividing. In places they were collected into groups of from ten to twenty, but they were not especially numerous around the capillaries. Dr. Hughlings-Jackson has since had another case of neuritis without coarse cerebral lesion, which he hopes shortly to place on record.

I think the view I have been advancing—the similarity of the changes in the optic nerves and brain—is of much practical importance, for we can see the optic nerves during life and, I submit, infer similar changes to what we find there in the brain. If this correspondence holds good, the ophthalmoscope may help us to correctly interpret many cases. Thus, in some cases of lead-poisoning we find optic neuritis. When such patients die, all the naked eye can discover is either atrophy of brain or no marked alterations. The test-tube tells us that the brain is saturated with lead. But neither the naked eye nor chemistry tell us what changes have gone on in the brain producing the headache, the delirium, the convulsions, and the coma, which have preceded death. Moreover, we know that under appropriate treatment the optic neuritis and nervous symptoms may coincidentally disappear, as in a case which I hope shortly to report in 'BRAIN.' I believe that the neuritis in such cases throws a light upon the character of the cerebral lesion, that the neuritis we see indicates a simultaneous and like affection of the brain, the neuritis being an extension process from the cerebritis, or the two like nervous structures being similarly affected by a common cause—the lead circulating in the blood. I believe that cerebritis will be revealed in such cases by microscopical examination. If these speculations are correct, it shows the extreme importance of the routine use of the ophthalmoscope, apart from any defect of sight, or even sign of nervous disorder. The eye has been happily called the window of the brain. By looking through it with the ophthalmoscope we may often see exactly what is taking place within the cranium, when without this window we should remain in ignorance.

## Correspondence.

### NOTE ON DR. J. HUGHLINGS-JACKSON'S CASE OF AUDITORY VERTIGO IN APRIL No. OF 'BRAIN.'

THE following sentences occur:—"Things (i.e. things in the room where he might be) began to pass to the right—he said 'from his left eye over his nose.' The same thing kept passing continually in this direction; *reappearing* on the left, and again and again passing to the right; he did not see them *coming back*."

"Each eye was partially and very slightly rotated to the right, in frequent jerks from left to right. . . . I was much interested in hearing him *volunteer* the remark about the mantelpiece, that 'it is going in this way' (he jerking his hand from left to right to show what he meant)."

"Several of my medical friends tell me, referring to my account of the paroxysm I witnessed, that the mantelpiece 'ought to have appeared to move to the left—in an opposite direction to that of the eyes.' That the eyes moved to the right, I am sure; and the patient always told me that things passed to the right."

In thinking over this moving image, it appeared to me that it was produced, not during the jerk or spasm, but during the time of recovery of the eyeball to a state of rest. After each jerk, the eye must move back to its original position before another jerk, and the image then produced would be apparently moving from left to right; that is, in an opposite direction to that of the eyes. The rapidity of the jerk would probably be so great as to prevent the formation of a definite image, hence he would not see the things "coming back."

Dr. Jackson does not say in his paper whether he considers the moving image produced during the jerk, or between the jerks; and as he says he does not see anything discrepant betwixt his report and the direction of the eyes, I suppose he believes that the image is

produced between the jerks ; whereas his medical friends think that the moving image was produced during the jerks.

JAMES TAYLOR.

18, Newgate Street, Chester, April 1879.

[My belief is that the apparent movements occurred during the jerks. Dr. Taylor's contrary suggestion is a very ingenious one, and no demonstration can be given against it by the facts of the case I have narrated, so far as they are stated. I believe it is now accepted doctrine that size, distance, and position is an affair of movement, and thus one would expect that displacement of objects in a case of movements of the eyes to one side would be in the direction of those movements. In cases of paresis of one external rectus objects are displaced in the direction of the movement only partly made, but strongly attempted.

J. HUGHLINGS-JACKSON.]



## Abstracts of British and Foreign Journals.

### ON METALLO- AND MAGNETO-THERAPEUTICS.

THE following pages are a continuation of my previous report on the same subject ('BRAIN,' January 1879), and are intended simply to keep the reader *au courant* of what is being done in the novel field of metallo- and magneto-therapeutics. The subject has entered now upon a new phase, that of physiological investigation; and, so far, the general remarks I ventured to make six months ago have been rather confirmed than otherwise by the results obtained. Heavy blows have been struck at many arguments of the "expectant attention" theory, and the burden of disproof laid at its door. It must be confessed, however, that the *rationale* of the phenomena observed is still veiled in obscurity.

Dr. Beard, of New York, has published some papers (collected as a pamphlet, "Experiments with living Human beings") in which he propounds the principles which ought to govern our reasoning from facts in which involuntary or subconscious life comes into play. The sources of error in such cases may arise from the various interactions of mind and body that are below the plane of volition and consciousness, either in the experimenter or his subject; from conscious or unconscious deception on the part of the subject; from collusion, intentional or not, of third parties; from chance and coincidence. The conditions of experimentation should be:—No repetition of the same experiment upon the same patient; no previous knowledge of the experiment by the subject; no feeling of any kind on the part of the subject towards the experimenter; no display likely to awaken expectant attention; control experiments of every kind.

Dr. Beard thinks that Charcot failed entirely to observe these restrictions, and that his results are phenomena of "trance" in no way ascribable to any electrical action or even cutaneous irritation.

Dr. Sigerson gives us ('British Medical Journal,' Feb. 1 and 8) an elaborate review of the whole question raised by the "recently reported phenomena in connection with Hystero-Epilepsy and Cerebral Anæsthesia." His object is to show that Prof. Charcot and the other eminent men who took part in the Salpêtrière experiments did not fall into the gross errors of observations ascribed to them by the "Expectant Attention" party.

1. Much stress has been laid upon the fact that hysteria abounds with sources of deception conscious or unconscious. To this it may be replied that experiments are most successful with patients when the hysterical state is least pronounced, and fail during the paroxysmal periods. It must also be noted that they fail in states of induced lethargy or subjection. On the other hand, marked results have been obtained in cases of undoubted organic lesion without a trace of hysterical symptoms.

2. Hysterical attacks may be produced through physical as well as emotional causes, but in neither case has expectant attention any necessary share in the result. For instance, it has been clearly shown that pressure, quite unexpected, has caused (and removed) paroxysms; and that the same pressure at other times entirely fails, however much the patient's attention may have been engaged in the experiment.

3. In organic as well as hysterical cases there occur crigenic regions, within the limits of which only can pressure call forth paroxysms. Clinical evidence, as well as observations on epileptic guinea-pigs, show expectant attention to have no share in the phenomena.

4. Dr. Sigerson points out that the Commission of inquiry took more trouble to guard themselves against error than they are generally credited with, and draws attention to the fact that if hysterical patients may occasionally be inclined to promote the success of the experiments, there is no lack of cases where they try to mystify the observer, and show themselves thoroughly averse to the repetition of the process.

A fact that makes the explanation of metallic action through the effects of imagination difficult is, first, that it was never known to the experimenters which metal was likely to act in any particular case; second, that in whatever order they were applied, the same always proved active; third, that in some cases, though the patient was under the impression that the active metal was being applied, no result followed if an inert one was deceptively substituted.

Dr. Sigerson remarks, as I pointed out myself ('BRAIN,' vol. i. p. 560) that it is hardly rational to ascribe to "expectant" attention phenomena not only unexpected, but of the possibility of which nobody would form any idea. He alludes to the transference of anaesthesia. The fact is that metals were avowedly applied with a view to relieve or cure the loss of sensation, and the occurrence of the phenomenon of transfer took everybody by surprise, both physicians and patients.

With respect to the *rationale* of metallic applications, Dr. Sigerson reminds us of the galvanometric observations of Regnard (see 'BRAIN,' Vol. I. p. 546) and the action of feeble galvanic currents.

With reference to magnets, it must not be forgotten that they need not touch the skin, and the patient's eyes being bandaged, they could have no knowledge of the presence or absence of the influencing agent. Yet systematic testing of sensibility showed that it never reappeared except in the neighbourhood of the poles.

5. Dr. Sigerson relates an experiment by Prof. Schiff which exactly fulfils the conditions laid down by Dr. Carpenter. A solenoid is placed upon the anaesthetic finger of a patient whose eyes are bandaged. An observer periodically tests the sensibility, whilst another makes and breaks the current behind a screen, unknown to all concerned. The periods of returning sensibility uniformly coincide with those during which the current circulated. Another experiment of Dr. Schiff's was as follows: A patient was made to breathe through a solenoid, the current being made and broken secretly. After a long period, the right anaesthesia was discovered to be disappearing: the current had just been made. On proceeding to test the left side for the expected transferred insensibility, it was discovered to everybody's surprise that no such transfer had taken place. This result was doubtlessly due to the mesial position of the solenoid.

Prof. Schiff stated that he had observed a case of intermittent coccygeal neuralgia where the contact of, or breathing through, a solenoid invariably brought on a fit of pain. At the same time he refused to attribute the whole of the effects produced to a physical action until similar phenomena had been observed in experiments on animals.

Prof. Schiff has published in the 'Archives des Sciences Physiques et Naturelles' (Geneva, 1879) a contribution to the study of the effect of solenoids on the nervous system. From his own and other experimenters' results he thinks that (in the lower animals

at least) the cortical motor centres are not directly motor but rather sensory, so that the disorders of motion which follow their destruction is due to the loss of sense of tact and position in the limbs so affected. Having then destroyed such centres in several dogs, and allowed some months to pass until the immediate effects of the operation had entirely passed away, he submitted the affected limbs, duly tested as to the anaesthesia present, to the action of a magnetising bobbin. In all the experiments it was found that after a longer or shorter time gentle irritation of the affected paw, such as stroking the hair, &c., was responded to by movements. This result, ascribed to a return of sensation, would sometimes persist for some hours, and could be obtained again daily by repeating the magnetisation. The reader will find a full abstract of Prof. Schiff's important paper in the 'British Medical Journal,' for April 26th, 1879.

Prof. Vierordt (*Centralblatt*, No. 4, 1879) gives the results of a series of experiments made with the view of testing the influence of metallic applications on frogs. The conditions under which he worked were as follows: having removed the cerebral hemispheres, he suspended the animal and stroked or pinched gently the fourth toe with an ivory forceps. In each case six series of observations were taken: in the 1st, 3rd, and 5th the normal irritability was tested; in the 2nd, 4th, and 6th, a plate of zinc (or lead) was applied to the frog. From 30 to 70 excitations were made in each case, and the results tabulated according to the result obtained: (a) movement in both legs; (b) movement in one leg; (c) no movement. Thus it was easy to compare the data, and the general conclusion was that with the application of the metal the effect of irritation was nearly twice as great as without.

The January number of the 'Journal of Anatomy and Physiology' contains a paper of Professor McKendrick's, entitled 'Observations on the Influence of an Electro-Magnet on some Phenomena of Nerve.' The method and results, which are highly suggestive and encouraging to further researches, were as follows:

The sciatic nerve of an ordinary frog-preparation was stretched across the poles of an electro-magnet, and the current alternately made and broken. It was found that there was sometimes a contraction on closure, and usually on opening. These contractions were probably due to an induced current generated in the nerve, and not to any derived current from the battery used, as shown by further experiments with a sensitive galvanometer. It was found, accidentally, that on touching the nerve with a copper wire held in

the hand, contractions ensued when this was done, whilst the circuit was broken, but did not whilst closed.<sup>1</sup>

The theory first assumed to explain this phenomenon was that it was caused by a derived current, due to imperfect insulation from the floor, and apparently was confirmed by the fact that when the operator stood on a glass-legged stool no contraction took place. But further experiments showed this to be erroneous, as the phenomenon did not occur unless, *cæteris paribus*, the nerve was not in direct (or indirect metallic) contact with the poles of the magnet.

Other experiments were instituted with the nerve placed on a glass plate, at right angles to the lines of magnetic force. Enough was observed to show that the nerve was affected, as occasionally the muscles contracted; but this point requires further elucidation.

Professor McKendrick promises us fresh communications upon this highly interesting subject, but for the present does not attempt any explanation.

In the January number of the 'Journal of Mental Science' Dr. Hack Tuke has published an account of his visits to the Salpêtrière. The phenomena he witnessed are similar to those described in my first report, but the remarks of the distinguished author of 'The Influence of the Mind on the Body' are of especial value. He points out that the argument against the reality of metallic influence from the fact that in some instances inert bodies have produced similar effects would be very much like denying the action of opium or jalap, because bread-pills are known to have produced purgation or sleep. Extracts from a letter of Dr. Vigouroux are given, in which attention is drawn to the systematically overlooked fact that, besides the experiments upon certain Salpêtrière patients who are used for public demonstrations outside the Salpêtrière and in non-hysterical cases, experiments have been daily repeated for more than two years on an enormous number of patients of both sexes, in ordinary practice and in the

<sup>1</sup> This fact reminds one somewhat of the unipolar excitation produced by the contact of one electrode connected, for instance, with an induction coil. The latter phenomenon, by the way, seems greatly to have exercised Dr. Sigerson, who, in the paper above referred to, described it as "a curious discovery" made by him two years ago. The human body (even when insulated) in presence of an induction coil may be looked upon as an infinitely great reservoir at zero potential. Each pole being alternately charged positively and negatively, it is evident that there will be currents passing to and fro between the body, and the pole of the coil with which it is in contact. The electro-magnet used by Dr. McKendrick was very large, and perhaps this is why no effect was produced by the insulated operator. The necessity of having the frog in metallic connection with the magnet tends to confirm this explanation.

most opposite conditions. Persons absolutely ignorant of the new methods, experienced the same sensations as those of the Salpêtrière, and employed the same terms to characterise them.

In a reprint of his paper Dr. Tuke adds some valuable facts supplied by Dr. Müller, of Graz, who repeated the Salpêtrière experiments under circumstances which rendered deception very improbable. Disks of wood, bone, cork, glass, marble were used in one case, among others, for test-experiments; but results never followed unless the active metal, tin, was applied. The usual phenomena of recovery and transfer in cases of anæsthesia, achromatopsia and contracture were observed, and a new fact noted, viz. transference of hemiparaplegia. The Austrian Medical Society investigated those facts and recognised their genuineness. They report further successes in cases of lesions both of motility and sensation.

In presence of such an array of independent witnesses it would be premature, Dr. Tuke concludes, without further experiments, to assume that the influence of metallic applications is solely ascribable to expectant attention.

A case of hysterical hemianæsthesia is described in the 'British Medical Journal' (April 26, 1879), in which the effects of metals and galvanism were tried. The loss of sensibility extended over the whole of the left side, including the special senses. On Nov. 15th, the patient and all around her being quite ignorant of the nature and object of this clinical method, her eyes were bandaged, and plates of copper, zinc, iron and lead applied on left arm, without any result. On Nov. 18th a bracelet of gold coins was similarly applied. The anæsthesia was marked. When removed six hours later, pricking and pinching the arm caused pain. The arm further improved by another application the next day.

*Nov. 21st.*—A current of 40 Leclanchés was applied first to the posterior and then to the anterior surface of the left thigh for five minutes. At first nothing was felt; but as the skin reddened, pricking became painful. No phenomena of transfer were observed.

*Nov. 22nd.*—Faradism having been applied by mistake, the patient was found next day to be again completely anæsthetic on left side. From that time the galvanic current was systematically applied to the body, limbs and face, and a gradual improvement was observed, the *anesthésie de retour* after each application never being as complete as it was before the electrification. This result, however, was only temporary, and the patient relapsed into a state of almost complete hemianæsthesia.

Dr. Anderson, of Edinburgh, relates ('British Medical Journal,' Feb. 8, 1879) a curious case of hystero-epilepsy, with aphasia, which he treated with gold applications externally, and chloride of gold and sodium internally. The fits and hemianæsthesia were considerably improved; but speech still remained in abeyance. The continuous current proved curative of the residual symptoms.

The author made some measurements of the patient's electrical resistance, and found it greatly increased—a fact which by the way entirely agrees with my experience in hysteria, and is but too frequently lost sight of. But it is surely going too far to conclude, from this one observation, to a "specific action" of the selected metal.

A case of metallotherapy is given by Dr. Fieuzal in the 'Progrès Médical' for January 1879. The patient, a girl of 17, complained of amblyopia and dyschromatopsia, with slight paresis of the internal recti. The other senses were found to be also affected. These symptoms, as well as right hemianalgesia and anæsthesia, became so marked as to prevent the patient from going about by herself. Iron, the bromides and hydrotherapy were all prescribed in vain. It was incidentally found that a gold disk restored sensation locally, but this indication was not followed up. An electrical treatment was followed, but to no purpose; in fact the anæsthesia became more marked on the left side as well. At the same time well-marked hysterical symptoms about the throat, &c., appeared. At this juncture Professor Charcot was consulted, and by his advice gold was tried *intus et extra*. The only result was that the patient lost her achromatopsia, whilst her acuity of vision rather tended to become worse. On exchanging silver for the gold plaques, however, a gradual improvement of the special senses soon became evident, along with restoration of feeling in the right side.

A curious case of hysterical blindness, under the care of Prof. Dujardin-Beaumetz and Dr. Abadie, appeared in the 'Gazette des Hôpitaux' (Nos. 55, 56, 1879). The patient, aged 16, had never presented the least sign of hysteria, and was perfectly regular. One night she complained of headache, and the next morning she awoke absolutely blind and anæsthetic. Without giving her any hint of what was being done to her, three gold coins were applied to the left temple. Under their influence there was a slight return of sight in the left eye, visual acuity reaching to 1-10th. Next day a magnet was applied to both temples; but the severe headache it produced prevented its being left long in contact; a slight return of vision ensued. Magnetic applications were repeated for two or three days, but they brought on a state of

lethargy such as to compel their cessation. Gold was then resorted to, *intus et extra*; and other metals successively tried, but without any considerable effect: visual acuity of both eyes 2-5, slight diminution of hemianæsthesia. Static electricity was then resorted to, and with marked effect. After the first sitting the patient could read; and soon both special and general sensation were completely restored. There still remained a tendency to sleep, with occasional fits of lethargy.

A case is related in the 'Gazette Obstétricale,' by Dr. Dupuy, in which metals are supposed to have relieved vesical spasm. The patient, a woman of 40, had suffered from these symptoms for years, and had been cured, apparently, by antispasmodics, &c. She, however, had a severe relapse, during which the pain produced by the passage of the catheter was such as to produce convulsions and loss of consciousness. Such was the horror of the patient for the operation, that she went without drink for two or three days in order to avoid the necessity of daily soundings. Nothing seemed even to relieve this agonising symptom; and Dr. Dupuy resolved to give the metals a trial, especially as the patient was suffering from spasms in other parts of the body. Gold was found to increase the convulsions, whilst copper, silver and iron removed them at once. Disks were then applied, and within an hour the patient passed water spontaneously, abundantly and painlessly. Whenever afterwards micturition did not occur normally, the application of the disks would call it forth, though the act was not always unattended with pain.

Dr. Landouzy ('*Progrès Médical*,' 4, 1879) relates a curious case of lethargy induced by the application of a magnet. The subject of this observation had just recovered from an attack of severe hysteria, and still suffered from meteorism and other slighter symptoms. Care was taken that she should be entirely unprepared, and her attention was sustained away from the manipulations by means of steady conversation. Two minutes after the application of the magnet to the abdomen, a slight tremor was noticed in the region of the right wrist and labial commissure, and she seemed to fall into a deep sleep. Shaking, pricking, &c., failed to rouse her, but on removing the magnet the identical tremors were observed, and the patient awoke, remembering nothing of the whole affair but the sensation of a cold body on the abdomen. Her eyes were then bandaged again and nothing done to her; no effect followed until the magnet was again brought near her arm. The same phenomena followed as before.



When the magnet was applied to the abdomen, her eyes being opened, nothing but the cold sensation was noticed. If now, however, her eyelids were closed the same lethargy followed. Opening the lids sufficed to awake her. The experiments were repeated several times. *No effect ever followed when a piece of non-magnetised iron was substituted.*

The conclusion of the case of hysterical contracture treated with electro-magnets, of which an abstract is given in the fourth number of 'BRAIN' (pp. 555, 556) appears in the 'Progrès Médical,' No. 8, 1879. The treatment was interrupted during August, on account of an attack of pneumonia. In September it was found that the left arm had lost whatever it had gained, and was paralysed. Dr. Vigouroux now tried a systematic course of statical electricity: the patient being charged and sparks taken from her arm daily for ten minutes. By the middle of January the arm had recovered its full vigour. As the treatment went on, the menses gradually reappeared, and the ovarian tenderness diminished steadily.<sup>1</sup>

*Pari passu* with these changes, it was noticed that it became more and more difficult to produce artificial contractures by means of the electro-magnet, until at last this became entirely impossible; a proof, Professor Charcot thinks, of the final return of the nervous system to a state of equilibrium.

A case of lead poisoning with hemianæsthesia, treated by magnets, is related by Dr. Debove in the 'Progrès Médical' for February 1879. The patient was a painter who had had two attacks of colic previously. He was picked up insensible in the street and brought into the Hôtel-Dieu, suffering from hemiplegia with anæsthesia on the left side. The electro-muscular contractility was diminished, and during the month of August he suffered from a series of attacks of pain, with periods of coma and delirium. He gradually improved, and in January the only symptoms remaining were loss of power and loss of sensation (including the special senses) on the left side. For this he had been faradised much, but in vain. On Jan. 12th a magnet was applied in presence of Profs. Charcot and Trélat. In a quarter of an hour it was found that the skin

<sup>1</sup> The therapeutical value of statical electricity in certain cases of hysteria seems to be greater than its almost universal neglect would justify. Besides Dr. Vigouroux's case and that of Dr. Dujardin-Beaumetz just mentioned, Prof. Erlenmeyer has also published one lately ('*Contrib. f. Nrvhlk.*' No. 1, 79), in which the superiority of the old plate machine over the modern batteries and coils, for a certain class of cases at least, was shown.

had become sensitive all over (excepting about the nose and the sole of the foot, where the anæsthesia proved particularly tenacious). The mucous membrane of the mouth remained insensitive. The left eye can count the fingers at 40 cm. and recognise all colours; but there is an annular scotoma.

The improvement continued. On the 6th day the patient can see the fingers at 60 cm. and the hearing was normal on the left side.

In the 'British Medical Journal' for Jan. 18, 1879, Dr. Wilks makes some "Remarks on Hemianæsthesia and its Cure." He contends that neither galvanic action nor mental influence can explain the effects of metals in hysteria; in fact that no explanation can be forthcoming until we are acquainted with the nature of nerve-force. In the same paper he gives the termination of the case referred to in 'BRAIN,' Vol. I. pp. 551, 552 where electricity as well as metals failed to produce any effect. The patient was since readmitted into the hospital, but no treatment adopted: she was systematically passed over at each visit. One day she stated she was better, and from that time rapidly progressed, leaving the hospital perfectly recovered.

Prof. Benedikt (*Wien. Med. Presse*, Jan. 26, and *Berl. Klin. Woch.* 17, 1879) points out the importance of urine-testing in cases of hysterical spinal irritation. After the therapeutical indications derived thereby have been attended to, galvanism, or metallothrapy, in the shape of chains of zinc disks along the spine, are beneficial. He has also found magnets effective when applied along the cervical spine or even the limbs. The latter may induce a cataleptic state (also obtained by placing the hands over the closed eyes) which is followed by an abatement of the symptoms for several days.

In his Graduation thesis on "the Bilateral Functions in their relation to Metallothrapy" (Berlin, 1879) Dr. Adler concludes from his observations on *two* hysterical patients that the metallic applications act merely by their mechanical power of irritating, and this because a strong mustard-poultice was found to restore sensation and produce the phenomenon of transfer. On the other hand experiments on *five* healthy persons showed that metallic applications had no effect on sensation in some cases, either diminished or increased it in others; whilst a mustard-poultice always increased it at the point of application and diminished it at a corresponding spot on the opposite side. Based upon these scanty and conflicting data, the author apparently wishes to refute

the conclusions of the French Commission. Granted the "mechanical power of irritation" of metallic disks, what about the action of solenoids, &c., at a distance?

The author of an article in the April number of the 'Birmingham Medical Review,' after giving an account of the main experiments and opinions on the subject of metallic and magnetic agency, concludes, that expectant attention cannot account for everything. It does not explain, for instance, the phenomenon of transfer; nor the disappearance of achromatopsia with the same order of colours in every case; nor the fixation of the effect by superimposition of metals, &c. The author very properly invites his readers to preserve an attitude of "expectant attention" towards these observations which, whatever may be their practical value, are of high scientific interest in reference to the influence of metals and magnets upon the human organism.

A. DE WATTEVILLE.

### On the Cephalic Ganglion and Retina in the Arthropoda.

EMIL BERGER. (*Untersuchungen über den Bau des Gehirns und der Retina der Arthropoden*, § 5, 48. V. Tafeln. Wien, 1878.) By the employment of translucent sections the author of the above paper has investigated the structure of the cephalic ganglion-like masses—which he regards as the brain, in a series of members of the Arthropoda, making the demonstration of their analogy [homology?] to the central nerve system of the vertebrata his main object. It results too, from Berger's observations, that many relations, such as we may recognise among the invertebrata, are capable of opening out to us new points of view as to the structure and functional significance of certain cerebral regions in the higher classes of animals, and above all others this seems to hold good of the central organ of vision. It would be impossible to analyse with due brevity and clearness the details of this most exhaustive monograph.

### On the Structure of a Microcephalic Brain. By T. V.

ROHON (Vienna, 1879).—The author of this memoir gives the details of his investigations into the structure of the brain of a microcephalic male child, aged fourteen days, which had been kept for several years in alcohol. The results are of especial value, inasmuch as the whole brain was cut successfully into sections fit for microscopical examination. It is impossible here to enter into

elaborate detail, but a few of the principal points only may be noticed.

The cerebrum consists of a single mass without division into hemispheres. On the surface there is only one transverse sulcus; and before it, the rudiments of three longitudinal sulci. There is no trace of a ventricle in the interior. The structure of the cortex does not differ appreciably from the normal.

The corpora quadrigemina and optic thalami lie freely exposed to view. The cerebellum is much better developed than the cerebrum, particularly the vermiform process.

In the interior of the cerebellar hemisphere the nucleus dentatus appears of altogether remarkable size, while the cortex can scarcely be considered as well developed throughout. Corresponding with the defective development of the cerebrum, the crus cerebri is also deficient. The nearer the sections approach the spinal cord, the more normal do the appearances become.

In this case there is an arrest of development which has affected the cerebrum at a very early period, almost at its commencement; while the other regions have become arrested between the third and sixth months of their development.

H. OBERSTEINER.

**On the Termination of Nerves in Striated Muscles.**—TSCHIRJEV (*Archives de Physiologie*, 2<sup>e</sup> série VI.) has employed a new method, suggested by Ranvier, for the investigation both of the centripetal and centrifugal nerves of striated muscles, of which the following are the chief results.

Ranvier's method consists in soaking the tissue in lemon-juice before submitting it to the action of gold chloride, a method which is said to leave the elements of the tissues unaltered. The tissue is placed in gold chloride solution for 10–12 minutes, and then kept in a solution of formic acid of 5 per cent. for ten or fifteen hours in the dark. Or the preparation is exposed to light for twenty-four hours in distilled water acidulated with acetic acid, and then decolorised by formic acid.

The mode of distribution of the nerves in muscle, so far as relates to their appearance and direction, agrees with that described by Kölliker and Odenius. In addition to the vaso-motor nerves, there is in muscle a considerable number of non-medullated fibres which have nuclei in their course, and which frequently form large-meshed plexuses.

They occasionally are seen to join medullated fibres, but more

frequently they join themselves to nerves composed only of two or three non-medullated fibres with large nuclei, slightly thicker than the former, and having a membranous investment. These nerves associate themselves with the medullated nerves, lose their proper sheath, and become lost to view. Frequently also non-medullated fibres are seen which plunge between the muscular fasciculi and disappear.

Tschirjew has used Ranvier's method to trace these nerves to their termination. He finds that they do not end in the fasciculi of muscle, which have no other nervous supply than those of the truly motor terminations.

But on detaching the aponeurosis there is usually seen a large-meshed network of non-medullated fibres. A more minute examination shows that this is not a true network, but an arborisation produced by dichotomous division of the fibres. The individual branches do not anastomose with each other.

The fibres of this arborisation do not possess myeline. They run separate, or run with others similar in a trunk invested by a sheath.

It is easy to show that these fibres do not reach the aponeurosis until they have traversed the whole thickness of the muscle.

The secondary fibrils become more and more fine, and lose themselves in the tissue as very delicate fibrillæ, which in general terminate abruptly, and sometimes present small enlargements like those in the cornea.

The conclusion is that the non-medullated nerve fibres in muscle end in the aponeurosis and are the only sensory nerves of muscle.

Tschirjew has also reinvestigated by the same method the mode of termination of the motor nerves. After reviewing the various researches in this direction, he describes and figures the results of his own researches on different orders of animals.

His conclusions are:—

(1.) The granular cushion with fundamental nuclei which exists in the motor plaques of certain species of animals does not form an essential part of the motor ending, inasmuch as it is not constant in the animal series.

(2.) The terminal arborisation only of the axis cylinder ought to be regarded as the essential part. It may become reduced, as in the tortoise, to a simple terminal knob, occasionally furnished with a nucleus.

(3.) There is therefore no essential morphological difference between the nerve-terminations of striated and unstriated muscle.

Hence the different mode of contraction of these two kinds of muscle cannot be attributed to the differences of the terminations of their nerves.

**The Tone of Striated Muscle.**—TSCHIRJEW (*Archiv für Anat. und Physiologie—Phys. Abth.* 1879), in his investigations into the nature of "tendon reflex," had arrived at the conclusion that muscles were connected with the central nervous system both by centrifugal and centripetal nerves, which latter enter the spinal end through the posterior roots.

The tendon reflex he found depended on reflex contractions of the muscle, conditioned by mechanical concussion of the nerves, situated at the junction of the tendon and proper muscular tissue. In his histological researches ('Comptes Rendus,' Oct. 1878 *et supra*) he has found a rich plexus of nerves in the aponeurosis of muscles. These nerves at first accompany the intramuscular nerves, but leave them and end in the aponeurosis of the muscle.

The aponeurotic nerves he regards as the only centripetal muscular nerves, but they have no specific sensory function. The phenomenon of tendon reflex depends on irritation of these nerves. He has further inquired whether a certain degree of tension of the muscle, and thereby mechanical stimulation of the aponeurotic nerve plexuses, may not be the efficient cause of muscle tone. The fact of muscle tone has been denied altogether by some physiologists, but Tschirjew, by a series of carefully conducted experiments on the muscles of the rabbit, has shown that, under certain conditions of tension, the muscle becomes elongated on section of its nerves, and also that the curve of a muscle so separated from the centre exhibits elastic oscillations which are not found in muscles whose nerves are intact.

Against the hypothesis of a continuous muscle tone, upheld by Müller, Remak, Brondgeest, &c., it is urged that when the two points of attachment of a muscle are approximated, the muscle becomes quite flaccid. Hence the tone cannot be regarded as continuous, even when all the nervous connections are intact. The tonic contraction is manifested only when there is a certain degree of tension. The facts of ataxy, and anaesthesia without ataxy, are against the hypothesis of a muscle tone conditioned by reflex stimulation from the general sensory surfaces. We may have complete loss of cutaneous sensibility without muscular flaccidity, and vice versâ.

To the reflex tone conditioned by the irritation of the aponeu-

rotic nerves Tschirjew attributes the absence of elastic oscillations which otherwise would accompany every muscular contraction. On this absence of elastic vibration depends the graduation of our muscular movements. He ascribes the inco-ordination of ataxy to the absence of tendon reflex in the muscles and the consequent loss of reflex tone. In ataxy the centrifugal paths are intact as well as the mere elasticity of antagonistic muscles. The cause must be therefore in the centripetal part of the mechanism by which muscular tone is maintained.

When a group of muscles is thrown into action the tone of the antagonists is increased. Hence they offer greater resistance and prevent too great excursion or jerking of the levers moved, and stop the elastic oscillations which would necessarily occur if the resistance was dependent merely on elasticity.

In tabes, in which there is degeneration of the central paths of the centripetal nerves of muscle, or of their tracts of communication with the motor nerves, and hence flaccidity of the muscles, the movements assume a jerking character, and the limb is seen to make a series of wavering or fluctuating movements about the mark, when the patient is told to raise his leg to a given height and keep it there.

D. F.

**Note on Infantile Paralysis.**—Sections of spinal cord from three cases of Infantile Paralysis were shown at a recent meeting of the Pathological Society of London. These cases not only fill up a lacuna in English work on the subject, but I believe give valuable suggestions as to the progress of this disease, and merit special notice in this Journal.

It has been contended by the French pathologists—Charcot, Vulpian, and Joffroy—that the changes found in the spinal cord, *other* than those in one or both anterior horns, are due to secondary horizontal extension from the lesion in the anterior horn or horns.

The comparison of these cases will, I believe, show that quite another doctrine is tenable.

(1) Dr. Turner's case was as follows: A child, aged  $2\frac{1}{2}$  years, six weeks after the onset of infantile paralysis, was taken ill with measles, and died from broncho-pneumonia.

In the grey matter of the spinal cord the maximum of change was certainly found in the anterior horns—more on one side than on the other. But there were extensive changes in the posterior horns, and also in the antero-lateral columns. The changes con-

sisted chiefly in destruction of ganglion cells and infiltration of leucocytes around the vessels, and in groups throughout the grey matter: in fact, there was red softening. Moreover, there was distinct though early sclerosis of the antero-lateral columns, more on one side than the other. There seemed no reason to doubt that many of the changes were contemporaneous, and the important point to note is, that the area of PRIMARY disease extended far beyond the anterior cornua, although there, no doubt, the disease was at its maximum.

(2) In Dr. Taylor's case, twenty months had elapsed after the onset of the paralysis. There was extreme diminution in *all* the measurements of one side of the cord—the diminution being most marked in the anterior horn, where there was almost complete absence of the large motor cells.

In the other half of the cord there were some changes in the central and outer parts of the anterior cornu.

The French interpretation of the conditions found would doubtless have been that the changes, other than those in the anterior horn, were slow, progressive, secondary by horizontal extension from it. But in the light of Dr. Turner's case, it seems fair to ask whether they might not have been the results of an intense acute lesion affecting *primarily* the portions of the cord which were found altered?

In Dr. Humphrey's case, the child died twenty-five months after the period when the paralysis came on.

The changes found were limited to one anterior horn, and consisted almost entirely in disappearance of groups of the large motor cells.

Here it is reasonable to suppose the original lesion was less in extent and intensity. The absence of disease beyond the anterior horn would, at all events, show that there is not *necessarily* horizontal secondary extension, and would be compatible with the view that, if in any given case such extensive disease is found, it may well have been the vestige of the original lesion.

THOMAS BARLOW.

**Heredity in Epilepsy.**—In the *Archives of Medicine* (vol. i. No. 2; New York: April 1879) Dr. Langdon Carter Gray reports the case of an American lady, aged 40 and married, who was subject to epilepsy, and who transmitted this malady to all her children, nine in number. When twelve years old, she began to have paroxysms in which faces of demons were seen at the right side of



the right eye, causing a great feeling of terror; and up till her twenty-third year she continued to have seizures of this kind, about three times a week. Then epileptic fits of an ordinary character set in, and increased in frequency until they occurred twice or thrice weekly. Since she reached her thirty-fifth year she has had fits only at her menstrual periods. She has borne nine children, four girls and five boys, all of whom have died in convulsions. The effect of the maternal disease upon the children has become more marked as time has gone on. The first three children lived longer (one reaching the age of thirteen months), and had less violent and frequent convulsions. The next four died soon after birth, and had convulsions continuously; the last two lived only for a few hours after delivery, dying in coma.

J. CRICHTON-BROWNE.

**On the Influence of Brain Work on the Development of the Cranium and the Brain.** (*Gaz. des Hôpitaux*, March 13, p. 243.)—MM. Lacassagne and Cliquet have examined, by the aid of the *Conformateur*, the heads of 190 Doctors of Medicine, 133 rudimentarily educated, 90 illiterate and 91 prisoner soldiers, with the following results:—

Diameters.	Doctors.	Soldiers.		
		Educated.	Uneducated.	Prisoners.
Longitudinal . . .	85·29	81·97	79·13	81·10
Frontal . . . . .	48·91	43·65	42·35	41·12
Parietal . . . . .	52·58	49·66	50·27	49·90 .

There is thus a considerable difference in favour of the doctors, and this is especially marked in the frontal measurements. Moreover, the two sides of the head are not symmetrical—in the educated the frontal region is more developed to the left; in the uneducated the occipital region is more developed to the right. The head is larger (more developed), in the case of the educated than in those of inactive intelligence. Among the educated the frontal region is more developed in proportion than the occipital—and, if the difference is greater in the occipital, it is very trifling; while among the illiterate it is considerable.

**On the Habitual Tendency of Individuals to Direct Themselves to the Right or Left.** (*Ibid.* March 18, p. 253.)—

M. Delaunay has made a communication on this subject to the Société de Biologie, and classes his observations under the following heads.

*Race*.—M. Delaunay is led to believe that the lower races who generally write from the right to the left incline more to the left than the right, while the higher races direct their course to the right. (This has been observed by *cicerones* in public museums, &c., and is also upheld by the fact that the general rule is to keep to the right on the pavement and similar circumstances, evidently based on the tendency of the majority. One always deviates to the right of a mark when the eyes are shut.)

(M. Delaunay thinks that this tendency to go to the right has played a part in the great migrations of nations. The ancients, who took their bearings looking towards the south, had the west on their right.)

*Sex*.—Women do not tend to the right so much as men. (A woman takes the left arm of her husband. In meetings where the sexes are separate, the females are on the left, the men on the right, &c.)

*Age*.—Children commencing to walk incline to the left, till three or four years old, when they take the contrary course. Adults generally go to the right, while, on the contrary, aged people incline to the left. Senile demented at Ville Errard Asylum have been found to promenade the airing courts with the wall on their left always.

*Constitution*.—Idiots and imbeciles in the same asylum tend to the left rather than to the right. Curable cases and general paralytics take the opposite course. (In Parliaments, literary and scientific reunions, &c., the left represents the party of progress, and the right the retrograde element!)

M. Delaunay believes that the tendency to go to the right observed in individuals advanced in evolution is owing to the left frontal lobe getting the upper hand of the right. He quotes Broca and Roque, showing that the left lobe is heavier, more rich in grey matter and more convoluted than the right; and Lacassagne and Cliquet, showing that the frontal region is more developed to the left than the right in the case of highly educated people. On the contrary, in the case of individuals less advanced in evolution (inferior races, women, children, and aged persons), the right frontal lobe is the chief.

On the other hand, in the case of a well-educated, left-handed, man who always directs himself to the left, the forehead is more

developed to the right than the left. Lastly, the two lobes are equal in those persons who tend indifferently to either side.

**Development of the Encephalon after Birth** (*Ibid.* April 1, p. 302).—M. Parrot has made a communication to the Society of Biology on the naked-eye appearances of the brain in the first year after birth. He has made 96 autopsies on children, for the most part from the age of two days to four months, and finds the brain a very imperfect and very slowly developed organ.

The anterior part of the brain is developed more slowly than the posterior, and this is in accordance with the early ossifying of the sutures in the latter region. In four-fifths of the cases he found the development of the right hemisphere earlier than the left. The cerebellum is also more rapidly developed than the cerebrum.

**On the Hereditary Influence of Alcoholism, and Epilepsy of Alcoholic Origin** (*Ibid.* April 26, p. 377).—M. Lancereaux has come to the following conclusions in regard to this important subject:—

Purely functional disorders are frequent if not constant among the descendants of drunkards. This is especially the case with hysteria, and convulsions from such simple causes as intestinal worms.

The effects on the intellectual and moral development are also commented on. It has been pointed out that the children of the workmen of Paris, who consume largely adulterated liquors, have, as a rule, a precocious intelligence, which seldom fulfils its early promise. Among other evils are noted convulsions, epilepsy of puberty, tubercular meningitis and arrested development of the intellect.

Drunkards hand down their depraved appetite to their children, and the tendency makes itself known at the various epochs of life—it may be at puberty, during pregnancy, or at the menopause. Other vicious habits are traceable to the same source.

Besides, there are organic *imperfections* and *changes*, it may be embryonic malformations, inflammatory lesions or atrophies of the nervous centres. But above all epilepsy is the most frequent result of parental alcoholism.

M. Martin has made a study of 150 epileptic patients at the Salpêtrière, and of 83 of these the family history has been traced. He has formed two classes.

1. Where the alcoholic tendency of the parents was certified.
2. Where it was doubtful.

The first class includes 60 patients, that is more than two-thirds.

These 60 had 244 brothers and sisters, 48 of whom have had convulsions, while 132 were dead in 1874, and only 112 were alive. Moreover, the greater number of those still living are still young, and several have had serious diseases of the nervous system.

In the second class 23 patients had 83 brothers and sisters; 10 have had convulsions, 37 were dead in 1874, while 46 were yet alive.

In the first group one-fifth of the children had convulsions, more than one-half died early; whilst in the second only one-eighth suffered, and the greater number are still alive.

Finally, 83 families, in which one or more members suffered from diseases of alcoholic origin, had 410 children; and of this number 108 (more than one-fourth) have had convulsions; and in 1874, 169 were dead, 241 lived, but 83 (more than a third of the survivors) were epileptic.

**Duality of Cerebral Operations** (*Ibid.* May 15, p. 445).—M. Luys has given a lecture 'on the duality (*dédoublement*) of cerebral operations, and on the rôle of the isolated activity of each hemisphere in phenomena of mental pathology. He has considered this subject anatomically, physiologically and pathologically, and the following are his conclusions:—

1. In the normal conditions of the working of the brain the hemispheres are endowed with a certain autonomy.

2. The left hemisphere is earlier in its development and attains a greater weight than its fellow—in general by 5 or 6 grammes.

3. Though the cerebral lobes, under certain conditions act synergetically, there are circumstances under which this does not occur. In spoken and written language the left hemisphere alone enters into action.

4. In playing musical instruments, especially the piano, cultivation creates artificial conditions of cerebral activity by virtue of which each lobe acts separately and independently, producing mental and motor operation—reading the music, marshalling recollections, using judgment, and making co-ordinating movements.

5. In the domain of mental pathology these natural aptitudes for

autonomic activity of each cerebral lobe are susceptible of much exaggeration. In the case of the insane the discrepancy of weight between the hemispheres is much greater than normal, and it is the right lobe which is the centre of troperic activity. Sometimes the difference in weight is as much as 25 to 30 grammes, and that too without destructive lesion.

6. In certain cases of insanity (*hallucinés lucides*) the co-existence of sanity and insanity gives a rational explanation of the integrity of one lobe and the morbid hypertrophy of certain regions of its fellow of the opposite side.

7. In a great number of psychopathic conditions, ungovernable impulses, alienation with consciousness, &c., the morbid states can have no other rational and true physiological explanation than a transient discord between the hemispheres—one acting irregularly, the other normally.

8. With regard to the prognosis of mental disease—the survival and persistence of lucidity implies an integrity of one lobe with all its dynamic capabilities; and reciprocally the absence of this lucidity implies the simultaneous and parallel invasion of both hemispheres.

A. R. URQUHART, M.D.

### One-sided Epilepsy with Tubercle in the Motor Zone.

—Dr. ASSAGIOLI received a girl of ten years of age into the General Hospital of Venice (see *Giornale Veneto di Scienze Mediche*, Gennajo 1879), who was suffering from pulmonary and intestinal tuberculosis.

During the last days of her life there were convulsive fits confined to the right half of the body. The fits were principally in the leg, but also passed to the arm, neck, and face on the same side. During the attacks, which lasted five minutes, consciousness remained intact, the pupils were dilated. There were four of these convulsions in one day, and one, limited to the leg, two days after. From this till death, eleven days after, there were no more fits.

It was supposed that the lesion was in the cortex cerebri, though the possibility of its being in the ganglia at the base of the brain was not excluded.

On opening the skull there was found hyperæmia of the pia mater, and some irregular pieces of cheesy matter covering a surface of a square centimetre, infiltrating not only the superficial grey substance of the paracentral lobule, but also the underlying white

tissue. The brain substance round about was softened. The rest of the brain was healthy. There were pleuritic adhesions in the chest, crude tubercles in the lung and caseous degeneration of the mesenteric glands. Tubercular ulcers were also found in the intestines.

**Una Microcefala:** Osservazioni Anatomiche ed Antropologiche del Dottore CARLO GIACOMINI, con quattro tavole litografate. Turin, 1876.—Dr. Carlo Giacomini had an opportunity of examining the body of a microcephale, called Manolino, and comparing the skull and brain with that of two other microcephales, called Bertolotti and Rubiolio. The result of his studies is given in an octavo pamphlet of 90 pages, illustrated by some very fine lithographs of the brain of Manolino and the skulls and cranial outlines of all the three microcephales.

Dr. Giacomini, who has made a careful study of the literature of the subject, enumerates most of the cases of microcephaly which have been published of late years. Manolino died at the age of 17 years. She was 64 inches in height. The head was 4·10 millimètres, about 16 inches in circumference, and the weight of the brain was 550 grammes. This, of course, is not an extreme case of microcephaly, since brains of half that weight have been described. The weight of Bertolotti's brain was 323 grammes.

The intellect of Manolino was very defective, but on this point the details are scanty. The convolutions were broad and simple, and the occipital lobe did not entirely overlap the cerebellum. There was nothing Simian in the type of the brain, and a very careful dissection of the body showed that there were no anomalies which could be traced to reversion of type. The author believes that he was the first to make a careful dissection of the body of a microcephale; this, however, was previously done by Bischoff, in the case of Helena Becker. The results of Giacomini's inquiry confirm those of the German anatomist. Microcephaly cannot be considered an example of atavism, and the only thing Simian about the brain of a microcephale is its diminutive size. At the end of the pamphlet there is a stereographic profile of six skulls, beginning from the outline of a large head, and ending with that of Rubiolio, which is much the smallest of them all.

# B R A I N.

OCTOBER, 1879.

## Original Articles.

### SUNSTROKE AND SOME OF ITS SEQUELÆ.

BY SIR J. FAYRER, K.C.S.I., LL.D., M.D., F.R.S.

UNDER the designations of sunstroke, coup-de-soleil, heat apoplexy, heat asphyxia, thermic fever, ardent fever, insolation, and others, are included certain pathological states, which though differing from each other materially are not unfrequently confounded.

1st. There is simple syncope from exhaustion caused by heat.

2nd. A condition analogous to shock, due to the action of the direct rays of a powerful sun on the brain and cord; the nerve-centres, especially the respiratory, are affected; respiration and circulation rapidly fail, and death may result; recovery is frequent, though not always perfect.

3rd. Overheating of the whole body, blood, and nerve centres, either from direct exposure to the sun's rays, or more frequently, to a high temperature out of them; causing vasomotor paralysis and intense pyrexia; respiration and circulation fail, and asphyxia follows. Recovery frequently occurs, but is often incomplete, owing to structural changes in the centres, giving origin to a variety of symptoms indicative of lesions of a grave character, some of which are well illustrated in the cases subsequently cited.

The cases of simple exhaustion and syncope may occur during great fatigue or over-exertion, or when there is depression of vital power from any cause during exposure to a high temperature, as in the case of the stokers and engine-room men of steamers in the Red Sea or the tropics, when the temperature rises to  $120^{\circ}$  and upwards, in the vicinity of the furnaces where they are employed; or in the case of men, especially Europeans, in the tropics who are exposed to the intense heat and light of the sun's rays, which taking effect on the head, neck, and body, produce a condition like shock acting through the vagus and vaso-motor system. There is depression of nerve force and of muscular power; the skin is pale, cold, and moist, the pulse feeble. Death may occur in this state from failure of the heart; but complete recovery more frequently occurs. Asphyxia and apnoea may come on after premonitory symptoms of depression and weakness, during exposure of the head and spine to the direct rays of a powerful sun, when the atmosphere is much heated, and the nervous energy is depressed by over fatigue, illness, or dissipation. The brain and respiratory nerve-centres are overwhelmed by the sudden rise of their temperature, respiration and circulation fail, the latter probably owing to inhibitory action of the vagus. When death takes place very suddenly during great excitement or exertion, and exposure to heat, it has been ascribed to rapid coagulation of cardiac-myosin.

This, however, though it *may* occur occasionally, is generally a post-mortem change; the heart's action being really brought to a close by heat, in the same manner as it has been shown by Claude Bernard and Lauder Brunton, that the effect of a very high temperature on animals is first to accelerate, and finally to stop the heart, and especially the ventricles, in a state of tetanic contraction.

Recovery, though frequently complete, is sometimes tedious, and occasionally imperfect, ending in serious impairment of health or intellect.

The symptoms of this form of sunstroke are those of sudden and violent lesion of the nerve-centres, unconsciousness, cold shivers, feeble pulse; all the signs of depression, terminating in death by shock; or fatal reaction may result, with a



variety of conditions pointing to injury to the cerebro-spinal system. In another class of cases there is ardent fever, the body generally, including the nerve centres, is heated intensely; this may occur quite independently of the direct action of the sun's rays. It comes on frequently at night, or in the shade, in a building or tent, especially in persons who are depressed by fatigue, bad air, over feeding, alcoholic stimulants and the consequent depression, want of rest, illness, and notably when the air is impure from overcrowding, or from insufficiency of cubic space.

The temperature of the body may rise to  $108^{\circ}$ – $110^{\circ}$ ; respiration and circulation fail; there is dyspnœa, hurried gasping respiration, great restlessness; pungently hot skin, sometimes dry, occasionally moist. The pulse varies; in some it is full and labouring, in others quick and jerking; the head, face, and neck are livid and congested; the carotid pulsation very perceptible; pupils, at first contracted, dilate widely before death. Coma, stertor, delirium, convulsions, frequently epileptiform in character, with relaxation of sphincter, and suppression of urine, these are the precursors of death by asphyxia, and it may be that there is cerebral hæmorrhage.

Such are the cases to which the term heat apoplexy is given; and a large proportion of the fatal cases among Europeans in India is so caused. Recovery may partially occur, to be followed by relapse and death, or secondary consequences, the result of tissue change, may destroy life or impair health and intellect at a later period. The premonitory symptoms of this form of the disease may appear some hours or even days before the dangerous condition just described supervenes. There may be general malaise, disordered secretions, profuse and frequent micturition, restlessness, insomnia, apprehension of impending evil, hurried and shallow breathing, precordial anxiety, gasping; giddiness, headache, occasionally nausea or vomiting, thirst, anorexia, feverishness, which soon amounts to fervent heat of skin; the surface may be dry or moist, the pulse varies; and these conditions gradually become aggravated and frequently are worse at night, when the patient passes into a state of unconsciousness and dies.

The symptoms point to a profoundly disturbed state of the

cerebro-spinal nerve centres, and to pathological changes in the organs whose functions have been so greatly disturbed.

Death is caused by asphyxia and apnœa, in some cases probably by cerebral hæmorrhage. Recovery is often incomplete, resulting in permanent impairment of health, and generally in intolerance of heat and of exposure to the sun. These morbid conditions being due to heat alone are liable to occur whenever there is exposure to a high temperature, whether solar or artificial. Soldiers marching or fighting, when oppressed by weight of clothing or accoutrements, are apt to suffer either from simple heat exhaustion or from that form of insolation which results from direct action of a powerful sun on the head and spine. This is common enough in India and elsewhere during the hot season. It is not unknown in Europe, even in England during the dog-days. Soldiers, labourers, artificers, and people in factories, heated rooms, hospitals, barracks, tents, and even ships, may suffer from heat exhaustion, which may pass into the same dangerous condition of heat asphyxia. People in the hay-field, or otherwise exposed to great heat in this country, especially if they have indulged in excess of alcoholic stimulants and food, may suffer.

Men serving in the engine-room of steamers in the tropics—the Red Sea for instance—are often brought on deck in a state of complete exhaustion, from which they generally recover under the reviving influence of the cooler air, a douche of cold water, or a stimulant; weak persons with defective hearts may die in this state of syncope. Soldiers, or others, when exposed to great heat, may drop out of the ranks, fall in a state of syncope, and die on the spot, or pass into a state of coma and die later; or they may recover, after being in great danger, with damaged nerve-centres, and are rendered quite unfit for further service, or even residence in a hot climate. These cases occur on exposure to the direct action of the sun's rays when the atmospheric temperature is also high, and especially when unusual exertion is made, or when the individual is depressed by previous illness or the exhaustion due to dissipation, intemperance, or even undue indulgence in stimulants.

But the most serious cases are those that come on under

cover by night as well as by day, and apart from the direct solar rays. A form of disease described in India as ardent fever is of this character, supervening on the ordinary phenomenon of ephemeral fever. Heat alone, especially when the atmosphere is loaded with moisture so as to prevent evaporation from the person, is the real cause of the disease. Malarious and hygrometric conditions have no special influence beyond that which they may exert on the general vigour of the constitution, thus predisposing him to suffer.

The dry atmosphere of Upper India, with its hot winds, is much better tolerated than the damp atmosphere of Lower Bengal or Southern India, though the temperature is lower. Hot dry air favours evaporation, and thus keeps the body cool, whilst in the damp air, as evaporation is diminished, the natural cooling power is greatly diminished.

Vigorous, healthy persons of moderately spare frame, with sound viscera, and who are of temperate habits, if the atmosphere be pure and moderately dry, can sustain a great amount of heat. Acclimatisation has also some influence in conferring toleration. Fresh arrivals in the tropics are more prone to suffer than those who have become accustomed to the climate and have learned how to protect themselves. It is well known that a native can bear an amount of sun on his bare head and naked body with indifference, almost pleasure, that would rapidly prostrate a European. But when the temperature rises above a certain standard all succumb, and natives of India suffer and die like others in numbers every year from "loo marna," hot-wind stroke.

The extent and duration of the toleration of heat depend much on the vigour of constitution and actual state of health. The refrigerating powers of the body, when in health, enable it to support a very high temperature, considerably above that of the blood. Thus in the hot winds little inconvenience is felt so long as perspiration is free, but when that fails, suffering soon ensues, and the danger is great.

In the Fourteenth Annual Report of the Sanitary Commissioner with the Government of India, 1877, it is stated that 235 cases of heat apoplexy and sunstroke occurred in the army in India, of which 70 were fatal. The admission rate,

4·1, is almost the same as in 1876, and exactly the same as in 1865. The death rate, 1·22, is below the average of the last six years. There is nothing calling for special remark in the ratios of the different Presidencies as compared with their former history. Of the 235 cases, 189 occurred during the four months of May to August. The disease was widely spread. In proportion to strength there is no remarkable number of attacks at any one place. It gives rise to a fatality of 12·2 per cent. in the first and second years of the European soldier's service in India.

The deaths per 1000 strength :—

SERVICE.		
1 to 4 years.	5 to 7 years.	above 7 years.
1·48	1·05	1·50

AGE.		
Under 25.	25 to 29.	30 and upwards.
·65	·99	2·33

But of those who recover, or rather who do not die, many are permanently injured and remain invalids for the rest of life, which is frequently shortened by the changes induced; and the cases hereafter cited, for which I am indebted to Dr. Christie, the able superintendent of the Royal Indian Asylum at Ealing, will show what sad effects sunstroke may have on Europeans in India. These may be due to obscure cerebral or meningeal changes, which affect the sufferer in various degrees of intensity. Irritability, impaired memory, epilepsy or epileptiform attacks, headache, mania, partial or complete paraplegia, partial or complete blindness, extreme intolerance of heat, especially of the sun's rays, rendering a person otherwise fairly healthy quite incapable of serving in hot climates or of enduring any exposure to the sun; or, it may be, gradually ending in complete fatuity, dementia, or epilepsy,

perchance both: chronic meningitis, with thickening of calvarium, accounting for the intense cephalalgia; or in a lesser degree in disordered innervation and general functional derangement, which seriously compromise health.

In cases where death has occurred suddenly, as from syncope or shock, there is no very remarkable morbid change. The heart may be firmly contracted—it has been found in animals that died from exposure to a high temperature, when the blood and nerve-centres were heated to  $110^{\circ}$ ,  $112^{\circ}$ , or higher, that the heart was tetanically contracted—but not always so, for it is often flaccid. The lungs, brain, and its membranes may be congested, but not invariably; they are sometimes quite the reverse. As in cases of shock, the venous trunks, especially those of the abdomen, and the right side of the heart itself, may be filled with blood. The blood is dark and grumous, often imperfectly coagulated, and effused in patches of ecchymosis, rendering the body rapidly livid. The coagulability of the blood is impaired, and it is deficient in oxygen.

In death from ordinary cases of thermic fever (insolation), the lungs and pulmonary system are often deeply congested; the heart is firmly contracted with coagulation of myosin, and the whole venous system is engorged. The body, even before death, may be marked by petechial patches and extensive livid ecchymosis. The blood is generally more fluid and grumous than natural, and it may be acid in reaction. The globules generally present no abnormal change in form, but are sometimes crenated and have a diminished tendency to form into rouleaux. The body for some time after death retains a high temperature. When first opened, the viscera and interior feel pungently hot, and the incisions drip dark blood. Rigor mortis comes on very rapidly. The brain and membranes may be congested; in some cases there are evidences of cerebral hæmorrhage and serous effusions in the ventricles. But the disease is essentially asphyxia, not apoplexy.

In cases of simple exhaustion, remove the person to a cooler place, if possible. Give a douche, but not too prolonged, or it may over-depress. A stimulant may be useful; rouse, and

gently stimulate ; remove tight and oppressive clothing. Treat as in ordinary syncope—ammonia to nostrils, &c.

Rest, and avoid exposure to over-fatigue or to great heat.

In the form of sunstroke where the person is struck down suddenly by a hot sun, remove him into the shade, and allow a douche of cold water to fall from a height on his head and body, from a pump (or from a mussuck in India), or other similar contrivance. This should be freely resorted to, the object being twofold : to reduce the temperature of the overheated centres, and to rouse by reflex action. During the assault on the "White House picket," at the capture of Rangoon in 1853, numbers of men were struck down by the fierce April sun. They were brought to me, and laid out in rows, perfectly unconscious, *in their red coats and black leather stocks* (they wore them, in those days, even in action under a tropical sun). They nearly all recovered—for the time, at all events—under the influence of the douche, freely applied over the head and body. In some cases, rousing by flagellation with the sweeper's broom was added with great effect, especially in the case of Brigadier-General W., who I thought must have died. All, or nearly all, recovered, except two, both of whom had been bled on the spot, before I saw them.

In addition to the douche, stimulants, such as mustard-plasters, to various parts of the body, legs, abdomen, &c., and stimulating enemata which relieve the loaded bowels and at the same time rouse, may be useful.

When I say such cases recovered, I refer to the reaction at the time. In some there were consecutive symptoms of fever, cephalalgia, &c. ; and, were we able to trace their subsequent history, we should probably find that complete recovery never occurred. If recovery is incomplete, and followed by indications of lesion of nerve-centres, or of meningitis, other treatment of a more active character will be needed according to the conditions.

Future exposure to the sun should be carefully guarded against, and, unless recovery has been rapid and complete, the sufferer, if in India or the tropics, should be removed to a cooler climate, where he should be protected from all

excitement of mind or body, and the greatest care be taken not only to avoid all errors or excesses of diet, but also of stimulants.

In the graver cases of thermic fever, or heat asphyxia, heat being the primary cause of the disease, the object is to reduce temperature as speedily as possible and before tissue changes have been caused. As the hyperpyrexia is due not only to the direct operation of heat on the nerve-centres, blood, and tissues, but to the fever set up by vasomotor disturbance, remedies that may influence this disturbed condition are indicated. The result of the treatment in some cases seems to confirm the correctness of the theory. The use of quinine and of morphia by hypodermic injection have both been considered to produce good results by their influence in reducing temperature and blood pressure, and perhaps retarding tissue change. The quinine, I think, may do so ; I doubt the morphia.

Bleeding has now happily been abandoned except in rare and peculiar cases. The congested livid surface, the coma and stertor which formerly suggested it are not now so treated. There are cases in which it may be necessary in order to avert suffocation, but they are, I think, the exception. In cases where it has appeared at first to give relief and to mitigate the symptoms, the improvement has been only transient, and followed by relapse into a more dangerous and fatal condition.

I do not think any absolute rule in this or any disease can be laid down in regard to abstraction of blood. It is possible that there may be more danger to life in the labouring and distended heart and the embarrassed lungs, than in the loss of a few ounces of blood ; which, if it would have tided the patient over the danger, as I believe it sometimes might do, would be the lesser evil, and as such should be chosen. Each case must be treated on its own merits.

The treatment generally, consists in the judicious application of cold by affusion, or by ice, taking care not to reduce temperature too low. A thermometer in the axilla, mouth, or rectum, will keep you informed in this respect.

Great care should be taken not to prolong the cold application too far, as danger would attend continued depression

of the temperature below the normal standard of blood heat. The bowels should be relieved, and blisters may be applied to the scalp and neck, though I cannot but say I have not much faith in their efficacy. In the epileptiform convulsions that so frequently occur, the inhalation of chloroform may be useful, but the administration of it must be carefully watched. The earliest and most severe symptoms having subsided, the febrile condition that follows is to be treated on ordinary principles; the diet must be carefully regulated. As improvement progresses, symptoms of intra-cranial mischief may begin to supervene; where the indications are of meningitis, iodide of potash and counter-irritation, may be of service; removal to a cooler climate is essential. As a general rule it is desirable that the sufferer should not, for a long period at least, return to a hot climate, and he should be guarded against all exposure to heat, overwork, and anxiety of any kind.

The sequelæ of sunstroke are often very distressing, and render the patient a source of anxiety and suffering to himself and to his friends.

The less severe symptoms—those probably of the slighter forms of meningitis, or of cerebral change—occasionally pass away after protracted residence in a cold climate; they are, however, not unfrequently the cause of suffering, but of danger to, and shortening of life; pointing to permanently disturbed if not structurally altered cerebro-spinal centres.

The following cases, for which I am indebted to Dr. Christie, are very illustrative of the evil effects that, more frequently than is perhaps imagined, result from sunstroke.

“MY DEAR SIR,

“I send you, as far as I can learn them, short histories of seventeen cases of insanity following sunstroke. I believe the whole of them are uncomplicated with insobriety. As a rule, the previous histories are not very replete with information, and only in one or two cases have I therefore been able to go very fully into them.

“It appears that the attack in most cases was the same and of a violent character, the onset was of an inflammatory type from the symptoms displayed, gradually settling down into



imbecility; the few post-mortems gave confirmatory proof of this in the adherent and thickened membranes.

"The second case was interesting, as showing the violence of symptoms during the whole attack, and which appeared to arise from the pressure of the plates of the skull, not, however, altogether the result of the sunstroke, as there is evidence of injury when young; here a resemblance is observed in the case of Surgeon-Major M., in whose case the bony growths were hereditary, and the same violence of symptoms was exhibited. In no case was recovery complete; although improved, a certain amount of imbecility resulted.

"Looking at the result of the post-mortem, I am led to think that the worst of insanity after sunstroke is of an acute inflammatory character, attacking the membranes of the brain, and thus the grey substance, so that we find the mental symptoms more clearly defined than the physical; only in the one case of Surgeon-Major J. M. were the physical characters clearly marked, and in this case paralysis was permanent from the first onset of disease.

"How far an alteration of structure of bony growth may be traced to *coup-de-soleil* is a matter for further consideration; in all the cases I have had under care, the diploe has been found obstructed and the skull plates thickened, dense, and heavy, with, in two cases, distinct growth of bone; but both these cases are involved in their etiology, in the one case by an early injury, and in the other by a most decided hereditary taint.

"At the present time I have two cases under treatment, the one a case of acute mania, with partial recovery following *coup-de-soleil* in Bengal; the other a case that occurred last summer here in the country, not followed by actual mania, but an inability to use the brain as formerly, coupled with grave symptoms at the present time, such as headache, squinting, and incapability of fixing attention, coupled with sleeplessness. Of both of these cases I hope to get a complete history.

"Yours very truly,

"THOS. B. CHRISTIE."

## CASE 1.

“Gunner J. M., aged 71 years.

“Went to India in the year 1839, and was a steady, well-conducted soldier. On the 13th April, 1843, was admitted into hospital for coup-de-soleil, and was discharged as recovered May 29th following. He was readmitted on the 18th of June, complaining of pains in the head and abdomen, but was discharged again in three days. July 12th he was again admitted, having been drinking, and became very excited, but was discharged on Aug. 12th. This went on, till the excitement increasing, he was admitted into the asylum, Jan. 23rd, 1844, for hypochondria, but soon after showing symptoms of imbecility he was sent home to England. On board ship he became violent, on several occasions, attacking those about him and threatening to throw them overboard. He arrived in England, Sept. 1844, and was admitted into the Asylum at Hackney. Here his case ran through the usual course, resulting in dementia, with general incoherence of language and silliness of manner. He is a Roman Catholic, and many of his delusions relate to the fasts of the church, fancying e.g. that Saturday is a fast-day, and will not eat meat. He is still living, with symptoms of cerebral atrophy.”

## CASE 2.

“Ensign P. F. T., age 51 years at death.

“This gentleman when about three years of age fell from a table and received a severe blow on the back of the head; very soon after he appeared drowsy and was sick, and in the course of a few days symptoms of acute inflammation of the brain came on, and he became perfectly unconscious. The attack soon subsided under active treatment, and he recovered. At the age of 17 he went to India, and within two years was attacked twice with fever, and apparently slight inflammation of the brain; subsequently, when exposed, he was struck down with coup-de-soleil, from which in a few days he apparently recovered. After this his correspondence was noticed to be irregular, and the style of his letters incoherent and

rambling. His conduct partook of the same character, until decided insanity necessitated his return to England. He recovered considerably on the voyage, and resided with his friends for a few years, during which they noticed a gradual increase of symptoms, till his violence was so great that he was sent to the Asylum at Hackney, where he was admitted July 18th, 1845, suffering from acute mania. His symptoms continued, and he became noisy, violent, fretful, and took a great dislike to his mother and relatives. In Aug. 1870 he was transferred to Ealing, and continued to show the same symptoms. On July 5th, 1871, he complained of great pain in the hepatic region, and he was ordered a purgative; this did not, however, remove the pain, and in two or three days violent diarrhœa came on, which was not checked by chlorodyne, opium, &c., &c.; on the 14th his temperature was  $98\frac{2}{3}$ , and pulse 100; there was no tenderness, and the skin was acting freely. July 16th, temperature  $99\frac{2}{3}$ , but the pulse fell to 98, the diarrhœa continuing with blood; this went on till the 24th, when he died.

“Post-mortem revealed an enormously enlarged liver, and adherent to the diaphragm: on attempting to separate it, an abscess of the size of a cricket-ball burst, in addition to which the right lobe was found to contain six separate and distinct cavities. Intestines congested, villi distinct with ulceration of the glands, especially in the colon. Kidneys pale, but healthy. Spleen small. Right lung adherent, and a small abscess over the liver in the lower lobe, partially hepatized, and corresponding to the liver. Heart healthy.

“Calvarium dense and heavy; over right parietal bone an apparent bulging, and over the middle suture thickened; the result of early injury. Membranes thickened, arachnoid opaque, with patches of lymph, the result of old inflammation. Convolutions flattened, grey substance pale and very defined, brain generally firm but anæmic. Brain small and atrophied. Base of skull rough, with prominences of bony deposit, some being very sharp.”

## CASE 3.

“Gunner A. F., aged 26 years.

“Admitted into Colaba Asylum, Bombay, Feb. 22nd, 1845, after about two years’ treatment in hospital for coup-de-soleil; during that time he complained of headache, ringing in the ears, loss of sleep, and delirium; this went on till Sept. 1844, when he became violent, and shouting continually. On admission he was suffering from low fever and diarrhœa. Imagined people were talking and speaking to him, and that they were trying to force air into his bowels. The usual treatment was pursued, and he was sent home, arriving in England in May 1846. He improved considerably on the voyage, but still had delusions; these, however, soon disappeared, and he was handed over to the care of his friends in the following month of June. Nothing more has been heard of him.”

## CASE 4.

“Gunner R. M., aged 31 years.

“As far as can be learned, a sober steady soldier. Was admitted into Regimental Hospital July 25th, 1849, suffering from the usual symptoms of coup-de-soleil, from these he recovered and left; but was brought back in a few days from being found wandering about and talking incoherently; he gave sensible answers to questions, and there did not appear to be any suspicions as to drink. This went on, leaving the hospital for a short time, and being readmitted again, till he was sent to the Asylum at Fort William, and admitted there March 19th, 1851. He was treated with cold baths, purgatives, &c., and not improving was sent home to England and admitted to the Asylum at Hackney, April 30th, 1852, having sunk into a state of dementia, with general incoherence; he continued thus till he died from phthisis, on June 4th, 1856. Post-mortem revealed the calvarium very adherent to the membranes, the plates of the skull of unusual thickness, being at least  $\frac{1}{4}$  of an inch, with the diploe filled up; the arachnoid contained about 2 ounces of serous fluid; pia mater, opaque and thick. The substance of the brain was firm, but appeared healthy. The brain was small. Lungs studded with tuberculous matter.”

## CASE 5.

“Private J. F., aged 22 years.

“Believed to be a sober man. Was attacked with sunstroke, April 12th, 1852, and taken to hospital; he improved slightly under treatment, but gradually became much excited and subject to various delusions, imagining people were trying to kill him. On the 30th of April, 1853, he was admitted to the Lunatic Asylum, Madras, and after a short time sent home to England, arriving Sept. 14th, 1853, and admitted into the Asylum at Hackney; he was in a state of dementia and in weak bodily health; in the following January, viz. the 7th, he died from chronic dysentery. The result of post-mortem examination was as follows: calvarium dense; brain substance firm, and apparently healthy; membranes adherent, with about five ounces of serum within the arachnoid.”

## CASE 6.

“Private J. T., aged 30 years.

“This man had an attack of coup-de-soleil in Bengal, in August 1857, after which his mind was disordered, and he appeared to be suffering from general paresis. On the 13th July, 1858, he was admitted into hospital, and found in a state of amentia; was transferred on July 21st, 1859, to the Lunatic Asylum, Madras, and sent home to England, arriving April 10th, 1859, suffering from general paresis. The disease ran the usual course, and he died July 17th, 1860, but no post-mortem appears to have been made.”

## CASE 7.

“Private J. F., aged 30 years.

“A sober, steady man, had sunstroke at Moulmein in Oct. 1857, and was sent to hospital, where he was under treatment till Aug. 1858, and appeared to recover so as to be sent to duty. On the 9th January, 1859, he was again sent to hospital for irritability and strangeness of manner, refusing to do his duty, &c., and was then thought to be insane, though he did not appear to have any prominent delusions. Not improving,

he was, on the 2nd of March, 1860, sent to the Lunatic Asylum at Madras, and forwarded home to England, arriving on the 28th of June, 1860, and admitted into the Asylum at Hackney. He was suffering from chronic mania, and did not improve, dying on April 8th, 1861, from disease of the liver. Post-mortem examination:—The calvarium was thick, dense, and heavy; the diploe being obliterated. Dura mater thick, with opacity of the pia mater. The brain substance appeared firm; the grey matter varying from  $\frac{1}{4}$  to  $\frac{1}{2}$  an inch in depth. The ventricles contained a small amount of fluid, and a large number of cysts were on the choroid plexis; the convolutions seemed natural.”

## CASE 8.

“Gunner P. F., aged 29 years.

“Appears to have had sunstroke in 1859, and was admitted into hospital Nov. 12, suffering from cephalalgia. Complained of pain, principally at the occiput; he was strange in his manner as evinced by his dress, putting his belts on wrong, &c.; he improved for a few days, and then gradually became worse; but was kept under treatment till he was admitted into the Lunatic Asylum, Colaba, Bombay, on the 26th of August, 1860. He did not improve, and was sent home to England, arriving April 25th, 1861, and admitted into the Asylum at Hackney. He remained very silly, and on Sept. 16th, 1865, he was sent to his friends in Ireland, and nothing more has been heard of him.”

## CASE 9.

“Seaman, T. T., aged 22 years.

“Had sunstroke on the voyage out to India, and on arrival there was found to be insane, so was sent home to England, arriving Aug. 8th, 1863, and was admitted to the Asylum at Hackney, suffering from mania. He gradually improved, and on Feb. 16th, 1864, was sent home to his friends, and nothing more has been heard of him.”

## CASE 10.

“Private J. M., aged 27 years.

“Of sober habits and healthy constitution. Admitted into Depot Hospital at Benares suffering from acute symptoms, the result of coup-de-soleil. His brain was very active, and he suffered from violent delirium, requiring force to keep him quiet. After a few days this passed off, leaving him very low. His memory seemed affected, and although not violent, he was unmanageable and subject to occasional paroxysms of violence. There is no history of hereditary taint. Treated with cold affusions and tonics.

“Admitted into Fort William, Bhowanipore, Dec. 30th, 1858, suffering from sleeplessness, subject to despondency and paroxysms of violence. Treated by tonics. Sent home to England, and on ship’s arrival in Sept. 1859, was so far recovered as to be allowed to go to his friends.”

## CASE 11.

“Pensioner J. D., aged 40 years.

“Admitted into General Hospital, Sept. 1863, with the loss of the use of his right side after coup-de-soleil. From this he recovered and was discharged. After this he had a loss, and it appeared to prey on his mind, so that he was admitted into Bhowanipore Lunatic Asylum, Nov. 28th, 1863. Was violent and noisy, striking his head against the wall, attempting suicide; but after a short time he became quiet, and was sent to England, arriving April 26th, 1864, and admitted into the Asylum at Hackney, suffering from dementia and general paresis. The disease ran the usual course, and he died April 29th, 1867, but no post-mortem was held.”

## CASE 12.

“Lieut. R. M., aged 26.

“Had sunstroke at Arrah, but no particulars were sent with him. He was of temperate habits, and was admitted into Bhowanipore Lunatic Asylum, Dec. 9th, 1864, labouring under various delusions of a personal character. Was noisy and

violent, imagining his relatives ought to be shot for cowardice. He was sent home, arriving in England Jan. 28th, 1865, and admitted into the Asylum at Hackney. He remained much the same, and was removed by his friends Jan. 29th, 1866."

#### CASE 13.

"Lieut. C. M., aged 40.

"Had sunstroke, followed by insanity. No history of his case. Still living."

#### CASE 14.

"Lieut. B., aged 36.

"No history; but was a sober, steady man, and became insane after sunstroke."

#### CASE 15.

"Lieut. B., age 39.

"The same."

#### CASE 16.

"R. L., aged 22 years.

"Was out in the sun, shooting one morning; after exposure to solar influence, he returned home and soon after became insensible, with stertorous breathing and convulsions. No medical man saw him, and he was sent to Kyouk Phyoo Aracan, Burmah, for treatment. He gradually became conscious, with loss of power in the lower extremities. His general health gradually improved, but with loss of speech and defective intelligence. He was sent to England, arrived Dec. 24th, 1878, and was admitted into the Asylum at Ealing, suffering from dementia. He had recovered the use of his limbs, but his mental power was weak. He has improved considerably, but lately tonic contractions of the extensor muscles of right arm have set in, so that I am led to think there is mischief connected with the centres relating to the arm. Still under treatment."

#### CASE 17.

"J. M., aged 43, and a widower, Surgeon-major, Madras.

"Was much affected at the loss of his wife, and while at Pulney, Madras, in the year 1868 had coup-de-soleil from



direct exposure, followed by headache on the left side, difficulty of articulation, and other signs of cerebral disturbance; this was followed by singing noises in the left ear, and defect of vision of the left eye from dilatation of the pupil, followed by great contraction. The memory became defective of recent events, and an eccentricity of conduct and manner became evident. Exaltation of ideas, great excitement, and a loss of control, was followed by excess of depression. He was placed under appropriate treatment, but the symptoms of violence and aberration increasing, as evidenced in striking those about him, and his wish to purchase property on a large scale, he was sent home, and came under my care at the Royal India Asylum, on August 25th, 1870, suffering apparently from general paresis. His ideas were exalted and bombastic, facies unhappy and perplexed; tongue tremulous and quickly receding, pulse varying from 76 to 93; temperature also from  $96\frac{1}{5}$  to  $99\frac{3}{5}$ , gait unsteady and tripping. These symptoms continued till about August 1872, when there was a great improvement altogether; he became more rational and his walk more steady, so that he was enabled to leave the asylum, and resided for the following two years in Scotland. I saw him occasionally during this time; he lived temperately, and as far as I could learn had not shown any signs of venereal excess. He took interest in various things, and played golf, thus of course taking a large amount of exercise. On Nov. 5th, 1874, he was readmitted. At this time he is noted as suffering from the second stage of general paresis, very indistinct in speech, and labouring under extravagant and exalted delusions that he is to play a great part in India, that he is very rich, &c. &c. His pulse was small and quick, pupils strongly contracted, and temperature normal. Was treated with Ex. 1. Physostig. gr.  $\frac{1}{4}$  three times daily. The symptoms gradually increased in intensity, and the paralysis became more marked till the beginning of December 1878, when he took to his bed. From this time I believe contraction of the lower extremities set in, and the limbs became strongly flexed towards the chin; he became rapidly worse, and died Jan. 27th, 1879, from exhaustion; he took food freely to within a few hours of his death.

“Post-mortem examination as regards the head only, was made about sixteen hours after death, and the following observations made:—Rigor mortis persistent; body much emaciated; calvarium strongly adherent, the plates dense; diploe obliterated; membranes very vascular, thickened, and adherent to the surface of the brain along the median fissure: this was found on separation to be caused by three or four bony plates, of the size of a sixpence, with small spiculæ passing into the surface of the brain on the left side; the brain was smaller than usual, and weighed only 44 ounces; the grey matter was deficient, and the convolutions flattened, and apparently not so numerous. Nothing unusual appeared in the substance of the brain beyond very slight softening in the right thalamus opticus.

“I think from the short notes of the above case, as I said before, great interest occurs from the close resemblance at the autopsy of the facts observed in the brother’s case, plates of bone being there found as large as a shilling; the brother had never been in India, was not the subject of coup-de-soleil, and yet the disease ran somewhat the same course, but was of shorter duration. Venereal excess still may be to blame, but it must be remembered that he was well known as being a highly moral man, and did not mix in the society of those who would lead him into this form of dissipation.”

## “RE-EDUCATION OF THE ADULT BRAIN.”

BY J. MORTIMER GRANVILLE, M.D.

THE very interesting and important case narrated by Professor Sharpey in the April number of ‘BRAIN’ recalls one that fell under my own observation rather more than twenty years ago. I will state its principal features, without going into details, and then venture to make the two cases an occasion for a few brief speculations which I am desirous of laying before medical-psychologists, with a view to obvious practical inferences in respect to the treatment of what I conceive to be a not uncommon cerebral condition.

In 1858 I was requested to see a daily governess and teacher of music, who had been suddenly attacked with what was thought to be acute mania. I found a spare, somewhat angular, eccentric-looking young woman, *ætat.* 26, in a state of great excitement, hysterical and choreic. Within a few hours—after a paroxysm of considerable violence, during which she talked and sang wildly and was with difficulty restrained by those around her—she fell into a state verging on suspended animation, which lasted a week. The skin was cold, and presented a dark mottled appearance; the pulse was scarcely perceptible at the wrist; the breathing slow and seldom deep; there seemed to be complete loss of consciousness, and scarcely any trace of sensibility. The muscles were cataleptic, and the extremities dropped slowly when raised. It was barely possible to feed the patient by the mouth, by holding forward the larynx and placing the fluid far back in the pharynx with a spoon, when it seemed to flow down the *œsophagus* as through a flaccid tube. This condition, which was treated with the interrupted current from the occiput and nape to the

hypogastrium, and mustard-poultices down the spine, subsided very gradually. Then came the state I am chiefly interested to note. There had clearly been an exciting cause for the attack in religious excitement, acting on a nervous system exhausted by protracted toil as a teacher.

When consciousness began to return, the latest sane ideas formed previous to the illness, mingled curiously with the new impressions received, as in the case of a person awakening slowly from a dream. When propped up with pillows in bed near the window, so that passers in the street could be seen, the patient described the moving objects as "trees walking;" and when asked where she saw these things, she invariably replied "in the other gospel." In short, her mental state was one in which the real and the ideal were not separable. Her recollections on recovery, and for some time afterwards, were indistinct, and, in regard to a large class of common topics which must have formed the staple material of thought up to the period of the attack, memory was blank. Special subjects of thought immediately anterior to the malady seemed to have saturated the mind so completely that the early impressions received after recovery commenced were imbued with them, while the cerebral record of penultimate brain work in the life before the morbid state was, as it were, obliterated. For example: although this young woman had supported herself by daily duty as a governess, she had no recollection of so simple a matter as the use of a writing implement. When a pen or pencil was placed in her hand, as it might be thrust between the fingers of a child, the act of grasping it was not excited, even reflexly: the touch or sight of the instrument awoke no association of ideas. The most perfect destruction of brain-tissue could not have more completely effaced the constructive effect of education and habit on the cerebral elements. This state lasted some weeks, and the "recollection" of what had been "forgotten," to use conventional terms, was slow and painful, needing, or, as I would now say, seeming to require, a process of re-education as distinct as (though, I judge, less prolonged than) that which proved necessary in the case detailed by Professor Sharpey. In the end recovery was mentally and physically satisfactory.

I cannot assume that anything in these two narratives will strike the practical psychologist as novel, or of even unfrequent occurrence. The clinical aspect of such cases has been sketched times without number. Nevertheless they present features of interest, as viewed from an etiological stand-point, which may be worthy more than a passing notice.

Either of three conditions may, I believe, be set up by brain disturbance, or disease, causing "loss of memory":—1, complete destruction of cerebral cells; 2, withering or blighting, which amounts to obliteration of the cells without destruction of their nuclei;<sup>1</sup> 3, a suspension of function without arrest of nutrition, as though a particular area of the cerebral organism were thrown out of the circuit of energy.

In the first event there will be final effacement of the records of ideation. So far as the cells destroyed are concerned, they and their properties are lost for ever. If the functions previously performed by these strata or tracts reappear, it must be because some other part of the brain has taken up the business vicariously—as I believe is possible with nearly every function or manifestation of mental energy. In the second event, when the cells are withered but the nuclei remain, a new crop of cells may spring from the parent organism, and after a lapse of time sufficient for development, the educationary record will reappear, the seed reproducing its kind, plus the effect of training and ideation. It may be that there will need to be so much re-education as to cultivate the new growth, and perhaps a re-impression of purely objective ideas, but it may, and probably in the majority of instances does, happen that the new cells will be developed with all the characteristics of the old. In the third event recovery may occur instantly, almost at any moment, if the obstacle to communication is overcome or breaks down in convalescence, so that the isolated, but scarcely injured, congeries of brain cells may again be energised. I speak of brain cells instead of "nerve-molecules," because, even accepting the vibration theory, it must be assumed that the vibrating particles are cellular vital organisms.

<sup>1</sup> I use the term *nucleus*, here and throughout in a non-physiological sense, simply to designate the seat of life in a cell, whatever that may be.

Supposing the states I have described to exist, I venture to suggest that the development of a new crop of cells from denuded germs or nuclei will account for the facility with which re-education, in cases like that described by Professor Sharpey, reproduces *knowledge*, even at a period of life when it is not easy to learn. What the new training and teaching does is not so much to impart information as to foster the growth of a new crop from the old seed, just as an after-crop may be procured by breaking up an over-stocked soil and applying the stimulus of manure. It is always possible that in the first process of instruction more seed may have been sown than germinated. Some good mental seed doubtless falls on barren ground, and it is perhaps due to the vitality and subsequent germination of this seed, ideas we do not seem to have cultivated deepen as the years go on.

Meanwhile I fancy it is as the progeny of old nuclei the physical bases of a revived memory are restored during general recovery in cases of the class before us. It seldom happens that the re-educating process needs to be very explicit or prolonged. Far less teaching than would have sufficed to implant the knowledge originally will cause it to reappear. In cases where the cells only are destroyed and their centres of vitality remain, it may even happen that the mere establishment of health will suffice to bring about complete restoration. When the new cells grow, the old memories will be revived. This is what takes place in ordinary cases, when, although no especial pains are taken to re-educate, the "lost" knowledge returns. The completeness of the recovery will probably depend on the vigour of the first growth, and is doubtless governed by the same law which determines permanence or tendency to revert to an old type in the propagation of recently impressed or acquired qualities of species or family. Ideas, or an organic tendency to form particular conceptions, are certainly transmitted from parent to child. The cells first developed in a foetal cerebrum are probably imbued with the qualities and properties of the brains of the mother and father, in different proportions. The transmission of germs of mental character which slumber through one generation and awaken with all their ancestral energy in the next

is a recognised fact. It will therefore probably happen that the new crop does not at first present all the features of that which was blighted by disease, but develop part of its characteristics later on. Thus vigorous health at an advanced period of life will sometimes produce a perfecting of the recovery commenced, but not consummated, years before.

Cases of the first and third class are very likely to be confounded in practice. Final destruction may be assumed when perhaps, a tract has been isolated without being destroyed. In this way I venture to think hopeless dementia is occasionally diagnosed, when what has happened is the disconnection, or throwing out of the circuit of cerebral energy, of a particular tract or stratum of elements; and, unless watched, partial recovery, susceptible of treatment, may happen without being observed and helped at the critical moment.

Treatment for the first class of cases is valueless; for the second, the cure must consist in the reproduction of brain-cells, or rather, as I have suggested, the development of a new crop from the denuded nuclei of blighted cells. The so-called "re-education" is only in a limited and scarcely physiological sense educationary. It is a repetition of the training, not so much to teach as to stimulate the growth of new organic elements from pre-existent germs imbued with formative forces and characteristics which must themselves determine the physico-mental result. If new cells *are* produced they will be found already educated, that is, endowed with inherited characteristics which constitute the physical bases of memory. The educated germ naturally produces an educated cell. Upon this hypothesis rests the whole theory of heredity, species, and transmission.

In the third class of cases, recovery occurs as an accident of treatment, except when in the presence of a constitutional cachexia like syphilis, specific medication may remove the grip of disease which, so to say, holds the mental organism in fetters that its energy cannot act. It will, I think, be often found that the seemingly permanent losses of memory which occur after acute disease are due to the isolation of special strata of cerebral tissue by the stasis of syphilitic or gouty disease. Mercury, iodide of potassium, or colchicum,

may in this way serve as a "memory-powder," and work a cure.

The two points I am chiefly anxious to place on record, without any claim to novelty of suggestion are, first, that what is called re-education is often simply the fostering of a natural growth—never harmful unless overdone, but of less value than may at first sight be supposed—second, that, in the absence of special indications that what seems to be helpless dementia is actually what it seems, i.e. a physical destruction of brain-cells; it is always *possible* the patient may recover, and therefore never justifiable to write a case off as incurable, and leave it to drift unnoticed and unhelped.



## ON AFFECTIONS OF SPEECH FROM DISEASE OF THE BRAIN.

BY J. HUGHLINGS-JACKSON, M.D., F.R.C.P., F.R.S.,

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It will doubtless have struck all physicians as strange that Speechless patients should have such recurring utterances as jargon No. 1 (Vol. II. p. 205), or single words, No. 2 (Vol. II. p. 205), or, what is most striking, such as "I'm very well," "Come on to me," No. 3 (Vol. II. p. 208).

It may perhaps be well to give some examples of these several kinds of recurring utterances from Trousseau's admirable Lecture on Aphasia. (I take them from Bazire's translation for the New Sydenham Society, adopting his rendering into English of the utterances of real words from the French; some of the utterances were jargon.) One patient could utter nothing whatever else than "Oh! how annoying!" Trousseau says of this patient: "Although she looked intelligent and behaved like a sensible person, I could never obtain another word from her." Another patient uttered, "Vousi, vousi." Another at first could only utter "Monomentif"; later on a few monosyllables ended by "tif," or the first syllable of a word and "tif," instead of the second, as "montif," for monsieur. Another uttered "Pig, animal, stupid fool," and that alone. Another, "Nasi bousi, nasi bousi." Another, "My faith!" Another patient, "Cousisi, cousisi." (This patient, by the way, had the occasional utterance when very excited, "Sacon, sacon," which Trousseau supposed to be an abbreviation of "Sacré nom de Dieu.") Another, only "Oh! mad." Another, "Sacré nom de Dieu."

It will have occurred to the reader that these utterances are not in the same category as "yes" and "no," in regard to the hypothesis stated, Vol. II. p. 215. We cannot say that the cases in which they remain show a reduction to the most general of all propositions. Some of the utterances of group No. 3 are propositional in form, but yet not of use as propositions to the speechless man; they are to him not propositions at all; figuratively speaking, they are propositions intellectually dead; they serve only emotionally, and in that way subsidiarily, as a sort of articulatory material on which the voice may vary. "Yes" and "No," group No. 4, Vol. II. p. 209, also are occasionally from the mouth of the speechless man, intellectually dead (See No. 4 (a), Vol. II. p. 210), and serve only emotionally; nevertheless these utterances so serving are in a different category from those of group 3. We have even on this basis to ask, "How is it that some speechless patients retain an utterance of no propositional value to them, but one which to healthy people is a proposition of a *special* character, applying to a particular combination of circumstances, whilst other aphasics retain an utterance of no propositional value to them, but one which to healthy people is of propositional value of the most *general* character, applying to any combination of circumstances?" Of course (see Vol. II. p. 209) the utterance may be an elaborate (compound) interjection, as "Oh, my God!" but we prefer considering those utterances which in health are propositional.

The problem may be presented in one case. A patient has "yes" and "no," and has also some elaborate recurring utterance in propositional form. Supposing his "yes" and "no" to be utterable only emotionally (see No. 4 (a), Vol. II. p. 210), we may say that he has two intellectually dead propositions, one of a highly special kind, another of a very general kind.

We wish in this instalment to show that the retention of recurring utterances, other than "yes" and "no," although apparently is not really exceptional to the principle of Dissolution. In all cases of Dissolution there is supposed to be a reduction to the more automatic, more organised, to the earlier acquired of the processes, represented in the centres

affected. It is, I think, plain that this course is in some cases apparently irregular. There are conditions interfering with what we may designate the Simple Course of Dissolution.<sup>1</sup> Let me illustrate this by a case from another class of nervous affections. Just after a slight epileptic seizure there is Dissolution, which in this case is temporary loss of function of more or fewer of the highest of all nervous arrangements; the patient acts grotesquely; but his actions (occurring during energising of nervous arrangements next lower than those which have undergone Dissolution) are not always rangeable as being more organised, &c., than his usual doings; sometimes they are plainly largely determined by particular events happening just before the seizure—that is to say, there are conditions interfering.

The matter to be discussed is not of scientific importance as bearing on the Principle of Dissolution. The discussion will help us, I think, to clearer notions on the nature of Defects of Speech (our No. 1, see Vol. I. p. 314). Were recurring utterances alone in question it would not be worth a physician's while to consider them at much length. We shall in the next instalment consider other kinds of nervous disease in which we find phenomena analogous to the recurring utterances of speechless patients.

The following hypothesis is advanced, and for the present is applied to but one set of recurring utterances, the group No. 3, Vol. II. p. 208, and only to those of them which have

<sup>1</sup> The reader is asked again to observe that the term Dissolution is used, as it is by Herbert Spencer, for a process which is the reverse of Evolution. I am sometimes told that it is not a fit term to apply to the results of disease on the nervous system; granting that those results are the opposite of Evolution of the nervous system. I do not believe they can be shown to be the exact corresponding opposite. Even supposing that the term does not properly apply, I submit that it is inexpedient to coin another word for a process the reverse of Nervous Evolution, when Dissolution is already in use for the reverse of Evolution in general. Affections of speech are examples of Dissolution occurring in a subordinate cerebral centre. Insanity, as for example the case of post-epileptic insanity mentioned in the text, is Dissolution, beginning in the highest cerebral centres, in those centres which are supposed to re-represent all that the subordinate centres have already represented, and thus, indirectly, to represent the whole organism; the highest centres are the substitute of our highest, latest, ever changing mental states, the abstract name for which is consciousness, there being really a series of consciousnesses.

in healthy people propositional value. *Such recurring utterances as "Come on to me" were being said, or rather were about to be said, when the patient was taken ill.* Here we have, it is suggested, a condition interfering with the simple course of Dissolution.

In order that we may show this hypothesis to be reasonable, the reader must bear vividly in mind that "taken ill" means here the occurrence of damage in a region of but one half of the brain, the left usually, sufficiently extensive to cause loss of speech. He must keep in mind that the nervous arrangements in this cerebral region are *destroyed*; it will not do to think of the disease vaguely as something "disordering certain functions of the brain." And on the psychical side he must look on the condition as one of *loss of the words* used in speech. The expression loss of memory for words is too indefinite, or is only verbally definite. It is equally important for him to bear vividly in mind that the other half of the brain, the right usually, is not at all injured. He is asked never to lose sight of the fact that, although the patient is rendered Speechless by the disease, he is not thereby rendered Wordless. We must indeed bear most vividly in mind that *the patient has words remaining*; it will not do to think of this positive condition under the vague expression that "he retains a memory of words." If we do use such redundant expressions, we must be thorough in our application of them and say two things, (1) that the speechless patient *has lost the memory of the words serving in speech*, and (2) that *he has not lost the memory of words serving in other ways*. In healthy people every word is in duplicate. The "experiment" which disease brutally makes on man seems to me to demonstrate this; it takes one set of words away and leaves the other set. The speechless patient has *lost* that set of words which serves in speech; he *retains* another set of words serving in other ways;<sup>1</sup> he *retains*

<sup>1</sup> The use of such highly technical expressions as "memory," in "explanation" of complex symptomatic conditions, seemingly definite and authoritative, is largely to blame for our remaining with our ideas on nervous diseases out of focus. It is not always vividly realised that retention of memory of words can mean anything more than a retention of words, the word "memory" in that context being really surplusage. There is almost the idea that the speaker *gives* words to the persons spoken to; all the speaker does is to rouse words already

all the words of the (his) English language in so far as "receiving speech" of others goes; and for other purposes he has *lost* their duplicates by which he should speak. It is suggested that whilst the speechlessness answers to the damage in one half of the brain, the retention of other kinds of service of words answers to the fact that the other half is not damaged. Attention is also asked to the hypothesis already stated, and hereafter to be expounded at length, that every proposition in health occurring during activity of the left side of the brain is preceded by a revival of the words of it during activity of the right half.<sup>1</sup> This is stating the hypothesis roughly and inadequately; all that need be insisted on is that the unit of action of the nervous system is double the unit of its composition, and that, correspondingly, all mental action is dual; subjective followed by objective. But as roughly put the hypothesis will serve our present purpose, since the speechless patient has no left half of his brain so far as its use in words goes.

Here brief remarks may be made on dual action in other mental processes in order to make clear what is the nature of the dual service of words. If I do not succeed in showing that there is duality of mental operations and what is the nature of that duality, I shall fail to make clear my meaning as to the nature of Recurring Utterances and to interpret many other difficult parts of our subject. We take for illustrations Ideation and Perception; that is to say, "propositions of images," which are symbolised by propositions strictly so-called. This is truly a part of our subject. It is indeed an entirely arbitrary proceeding to separate the images symbo-

organised in the person spoken to. Any one will readily admit that the pain of a pin-prick is in himself, not in the pin; and every educated man admits that redness is in himself when he uses the ordinary language, "This brick is red." Why then should we speak of "retention of memory of words" by the aphasic when he understands what we say to him without, at any rate, using that expression as simply convertible with the expression that he retains words (physically organised nervous arrangements for words)? Whose words are those which occur in the so-called hearing "voices" of the insane?

<sup>1</sup> There are then, it is supposed, two services of words by the right half of the brain; the "reception" of words of others, and the reproduction of words which precede our own speech. These are the two ways other than speech in which words are supposed to serve. Doubtless at bottom these two are alike.

lised from the symbols of images; in what we shall call the "conflict" both are concerned. Nevertheless the artificial separation of them is convenient in brief exposition. It will suffice for illustration to take count of but one image, although two images must be concerned in every ideational or perceptive process and also in the stage preceding ideation or perception (ideation may not be followed by perception, but ideation must always precede perceptions).

It seems clear enough that the process which ends in ideation or perception is dual. *Before* we can see or think of a brick, before either ideation or perception can occur, that brick by actual presentation or by indirect presentation through association, must have roused an image in us. When we say we see a brick, all we mean is that we project into the environment, ideal or actual, the image which that brick has roused in us. This image arisen in us is the survival of the fittest image at the termination of a struggle which the presented brick has roused in us, is the end of the subjective stage; the further stage, or the second stage, is objective—it is referring the image already roused in us to the environment, actual or ideal. The brick is for us nothing more than what it has itself roused in us, so that, instead of using such figures as that "the mind impresses its own laws on sensations" we experience during contact with external objects, or "that the mind originates something additional to them," or "works them up into different shapes," or that it "impresses its own form on them," we should, I think, use the opposite figure that an external object acts on us and develops in us such as we are, *what it can*, we being at first passive and it active. I think it is plainly so in dreams excited by peripheral local excitations. And in reverse, cracks and marks in the burning coals make us see faces in the fire, rouse faces in us.

Returning to the dual service of words. If we coin the term *verbalising* to include the whole process of which speech is only the end or second half, we may say that there are in it two propositions: the Subject-proposition followed by the Object-proposition, the latter being called speech. Spencer writes ('Psychology,' vol. i. p. 162): "A psychological propo-

sition is necessarily compounded of two propositions, of which one concerns the subject and the other concerns the object; and cannot be expressed without the four terms which these two propositions imply." It is supposed that the subject-proposition is the "survival of the fittest" words in fittest relation during activity beginning in the right half of the brain, and that this survival is at once the end of the subjective and the beginning of the objective stage of verbalising. The subject-proposition symbolises an internal relation of two images, internal in the sense that each of them is related to all other images already organised in us, and thus it symbolises states of *us*; the object-proposition symbolises relation of these two images as for things in the environment, each of which images is related to all other images then organising from the environment; thus it stands for states of the environment; the two propositions together symbolise an internal relation of images in relation to an external relation of images. Thus the separation we have made into speech use of words and not-speech use of words may appear arbitrary; it is, however, convenient.

In one of the cases to be presently mentioned in the text—the patient who said "Come on to me"—there was *left* hemiplegia, and thus the inference is irresistible that his speechlessness was caused by damage in the right half of his brain. But as he was a left-handed man, his case is an exception proving the rule. It is admitted that there are cases of left hemiplegia with aphasia in persons who are not left-handed. Besides granting fully the significance of the fact that in the vastly greater number of cases loss of speech is caused by disease in the left half of the brain, the thing of infinitely greater significance is that damage in but *one* half can produce speechlessness; it is equally significant that damage in neither half, produces wordlessness.

In a few cases I can state the circumstances of the onset of the illness, which seem to me to countenance the hypothesis stated. The man whose recurring utterance was "Come on to me," and sometimes only "Come on," was a railway signal-man, and was taken ill on the rails in front of his box. A woman in Guy's Hospital could only utter "Gee gee"; she

was taken ill whilst riding on a donkey. Dr. Russell of Birmingham has published the case of a clerk, who lost speech and became paralysed on one side, after hard work in *making a catalogue*; this poor fellow could only say, "List complete." Sir James Paget a few years ago had under his care in St. Bartholomew's Hospital a man whose left cerebral hemisphere was injured in a brawl. This man could only say, "I want protection."

The hypothesis further unfolded but applied only to the above-mentioned cases for the present, is that the words of the recurring utterance had been revived during activity of the right half of the brain, when the destruction of that part of the left half occurred which caused loss of speech, that they constituted the last proposition, or rather that stage of verbal revival (what we have called the subject-proposition), prior to the last proposition (the object-proposition). Ever after they were meaningless or, figuratively speaking, dead propositions, remaining utterances of which we can only say they were on one occasion speech; on that occasion fitted to indicate then occurring relations of things. On the rails, or certainly at some juncture, "Come on to me" *meant* "Come on to me," ever after the illness it meant nothing.

The expression "on one occasion" is not to be taken literally as meaning that; for example, the man who uttered "Come on to me" never said that before he was taken ill on the rails. The presumption is that he had said it on many occasions, when trains were approaching his post. What I mean rather is, speaking now of the healthy, that there is no such proposition in a man's mind, excepting when he is saying it. On the physical side we would suggest that the nervous arrangements concerned during the utterance of any such proposition are not excited, nor ready to be excited, in the particular order answering to such proposition except at the time of uttering it. Such propositions are new speech. The "same" proposition is new or "latest" speech each time it is uttered, if the words or syllables of it are not, so to speak, "kept ready made up" in that particular combination; at any rate it is "latest speech" when specially applied at a particular time to indicate then occurring relations of things which *are*



*not fully organised*; it is then, otherwise stated, a voluntary use of words.<sup>1</sup>

From the most automatic or "oldest" or "earliest" speech, applying to well-organised external relations of things up to newest speech, applying to now organising external relations, or otherwise put propositions now in the making, there are, it is supposed, on the physical side, only degrees of independency of organisation in the sense that the nervous centres concerned have lines of different degrees of resistance to currents of different force, entering the centres at different points. Other things equal, the more automatically serving (the more organised) nervous arrangements are lines of least resistance. There is, however, in the case of very frequently used and very automatic utterances, such as "Very well," "I don't know," a near approach to independent organisation, and in the case of the most common interjections a very near approach to it. Such utterances constitute what I call old speech, or, in the case of interjections, they are verbal processes lower and earlier than true speech—utterances not "now making," but nearly, if indeed not quite, "ready made up." And at the other extreme, in the highest, newest, or latest speech, there remains for a short time after the utterance a slight degree of independent organisation of the nervous arrangements concerned; did there not, we should not know what we had just said, and could not go on talking consecutively on any subject. If this reasoning be correct, such a

<sup>1</sup> The very same "utterance" which has *become* automatic by being often used for symbolising frequently presented, and therefore well organised, external relations of things would be once more a voluntary utterance, if it were used on a new occasion, that is for symbolising a relation of things not already organised, but only now organising. The "highest," "latest," or "newest," speech (the now organising or voluntary use of words), implies clear preconception; clear preconception is a necessary element for voluntary, as distinguished from automatic, operations; in voluntary speech the prior reproduction of words constitutes the preconception. If a man utters, as applying to a new set of circumstances, the most automatic utterance he has, or if he utters anything "for the sake of uttering it," as when asked to do so, there is then a voluntary utterance, for then the operation occurs after clear preconception. Disease shows this not to be a fanciful dictum. As we have seen, and we shall later on give further examples, a speechless patient may be unable to *say* (to repeat) what he has just uttered. We do not affirm that the word "say" in this context means a language process.

recurring utterance as "Come on to me" represents not only the last proposition *en permanence*, but is to be looked on as on the physical side, a keeping up of activity (of a greater readiness to discharge), of certain nervous arrangements, which normally exist only temporarily and only on special occasions, in particular combination, and which in health go quickly into subordinate function, and soon out of function.<sup>1</sup>

Such a recurring utterance as "Come on to me" was about to be new speech; the words remained *en permanence* as a dead proposition; a proposition stillborn. We can now describe the case of a man who has the two sets of recurring utterance as that of a patient retaining a new combination of words, and also an old combination—or rather in the latter two monosyllables, each of which is equivalent to a combination of words.

The reason for thinking that the recurring utterances under remark were and continued to be utterances occurring during activity of the *right* half of the brain, is that the left half of the brain is so extensively damaged that the patient cannot speak; he has no left half for their utterance. To say that the disease "caused" these utterances a positive condition is absurd, for the disease is destruction of nervous arrangements, and that could not cause a man to do something; it has enough to answer for in leaving him unable to speak. The utterances are effected during activity of nervous arrangements which have escaped injury. This remark may seem a truism here, but in more complicated cases it is very common to hear of positive symptoms being ascribed to negative lesions—to loss of function of nervous elements. (See p. 316, Vol. I., On Negative and Positive Elements in Symptomatic Conditions.) It is common at any rate for disease to be thought of vaguely as something "*disordering* the functions of the brain." In the cases we are now dealing with, and in cases of defect of speech, and also in cases of insanity, the function of some nervous arrangements *is lost or is defective*.

<sup>1</sup> Practically out of function, so far as our present subject is concerned, although, obviously, there would be no new acquirements possible, and no memory, if some nervous arrangements after their use did not remain lines of less resistance than before.

But it is an error to ascribe such positive symptoms as the recurring utterances in speechless men, the erroneous words uttered by those who have defect of speech, and the hallucinations, &c., of insane persons, to negative lesions—to loss or to defect of functions. These positive mental symptoms arise during activity of lower centres or lower nervous arrangements which have escaped injury, and are only to be thought of as symptoms in the sense of being the fittest psychical states arising during slightly hyper-normal discharges of lower or more organised nervous arrangements which are *then* highest, the normally highest having lost functions.

It might be suggested that not all, but only nearly all, the substrata of speech were destroyed, and that enough nervous arrangements remained in the left half for just one utterance. Since, as implied in a foregoing statement, I do not believe that words or syllables have nervous arrangements in the sense that there is one little nervous centre for each syllable or rather single articulation,<sup>1</sup> and for no other, and since too I believe that each unit of every nervous centre is the whole of that nervous centre in (different) miniature, I should be as ready as most people to accept this explanation. But a certain quantity of nervous arrangements, implying a certain quantity of energy, is required for every operation. As the Recurring Utterance is, as a series of articulations, very elaborate, and as the syllables are clearly enunciated at any time, it is not credible that slight remains of nervous arrangements can be concerned during the utterance of them, especially when looking at the matter on another side we see that these remains do not serve the patient to utter any other words whatever, except perhaps “yes” and “no,” which I however believe to occur also during activity of the right half of the brain. The man who uttered “Come on to me” uttered it any time, with no effort, rapidly, and in fact just as well as a

<sup>1</sup> The reader will observe that nothing is said in the text which implies that a word *is* a nervous arrangement resented an articulatory movement. All that is suggested (see Vol. I. pp. 305, 306) is that the *anatomical substratum* of a word is a nervous arrangement representing one or more particular articulatory movements. The reader will observe, too, that we sometimes speak of words, and at other times of their physical bases, but we do not, it is hoped, confuse the two different things.

healthy person could utter it; yet except for this and "yes" and "no," he could utter nothing else. It is submitted that it is not possible that such an utterance was effected during discharges of remains of a nervous centre so extensively diseased, that is to say, not during discharges of any part of a half of the brain which was damaged so much as to cause speechlessness. It is more likely to be uttered during activity of the undamaged half.

We repeat, the patient may have not only such a recurring utterance as "Come on to me," but he may have also the utterance "yes" or "no," or he may have both these words. We have endeavoured to explain the retention of "yes" and "no," p. 214, by saying that the patient is reduced to the two most automatic, the two most organised, of all propositions. We have tried to show that in a sense the principle of dissolution is not contradicted by the retention of the elaborate recurring utterances under remark. There are, we have suggested, conditions interfering with what we may call the simple course of dissolution. The patient retains not only his most automatic propositions, but his last proposition, the then most voluntary proposition; the former sometimes, the latter always, ceasing to be propositions.

Let us now look more particularly at the anatomico-physiological side; that is, examine the utterance from the side of degree of organisation of nervous arrangements. We must bear in mind that, to speak metaphorically, all disease cares about is degree of organisation; it respects processes the more they are organised, caring nothing about important or unimportant. At present we can only speak in outline.

In estimating organisation in its bearing on affections of speech or on any other sort of defects from nervous lesions, we have to consider recency of organisation, as well as degree of organisation. It is well known that in failing memory recent events are soon forgotten, whilst old events are remembered; but this is not without qualification; the *most* recent events are remembered as well as the old; on the physical side, nervous arrangements just discharged remain for a short time in a state of slight independent organisation, rivalling that of nervous arrangements discharged when the person was young

and vigorous, or that of nervous arrangements often discharged.

That the nervous arrangements for the words of the recurring utterance (No. 3) are parts of the patient is a truism; that the words must one time have been revived in propositional form, that certain nervous arrangements must one time have been discharged in a particular order, is another. The hypothesis that the words were being revived at the time when the patient was taken ill seems to me warrantable. As they remain always easily utterable, and they alone utterable, since the illness, it is plain that certain nervous arrangements which were being discharged when the patient was taken ill, or at any rate which were on some occasion being discharged in a particular definite order, remain permanently in a state of dischargeability far above normal. For the patient not only does not speak since his illness, but whenever since his illness he tries to speak, the recurring utterance comes out or nothing comes out. Note three equally important things—(1) he has it; (2) he has no other utterance (except perhaps “yes” and “no”); (3) he cannot get rid of it. It is certain that the nervous arrangements for the recurring utterance have somehow arrived at what is in effect a high degree of independence of organisation. We can say, if the hypothesis put forward be true, that the nervous arrangements for the proposition *just organising* at the time of the illness, those for the words last spoken or about to be spoken, remain as well as *the most and always organised* nervous arrangements for the most automatic propositions, and have somehow achieved a degree of independent organisation greater than that of any other word-processes except “yes” and “no”; probably by repeated utterance after their first utterance. On this matter we shall speak later, when considering the phenomenon called by Gairdiner “barrel-organism.” At present the explanation given may seem to be merely a verbal one. We may consider part of the question at once. Repeating that the patient has the utterance, and that he cannot get rid of it, we restate the question thus. How is it, (1) that destruction of one centre (certain part of the left half of the brain) leaves in state of hypernormal dischargeability particular nervous arrangements discharging in another centre at the outset

of the illness, right half of brain, a lower centre? and (2) How is it that they do not go out of function, as they certainly would have done had not that destruction occurred? We say again that the destruction in the left half is not the cause of the recurring utterance; a negative state of nervous elements cannot possibly be the cause of positive nervous symptoms. Nor is destruction of some nervous arrangements the cause, or at any rate not the direct cause, of certain other nervous arrangements discharging and remaining in a state of hypernormal dischargeability. But plainly, if the man had not been "taken ill," he would not have had such a recurring utterance; he would have been able *not to utter it*, the nervous arrangements concerned would have gone out of function; but since his illness, he must utter it when he tries to speak, if he utters anything. I believe the solution of the seeming paradox to lie in this, that destruction of function of a higher centre is a removal of inhibition over a lower centre ('Principle of Loss of Control,' Anstie, Thompson Dickson), the lower<sup>1</sup> centre becomes more easily dischargeable, or popularly speaking "more excitable," and especially those parts of that centre which are in activity when control is removed. So to speak, these parts become autonomous, acting for themselves, just as parts of the spinal cord below a diseased point become autonomous in some cases of paraplegia. Thus the disease *causes* loss of speech; it *permits* the increased dischargeability of the right half. It is just as cutting the pneumogastric does not cause, but permits, increased frequency of cardiac beats. But, the simple course of dissolution being interfered with in the cases of speechlessness we are writing about, the activity of the centre manifests itself in the way it was temporarily in when control was removed. The greater excitability of a centre uncontrolled by removal of its higher centre is supposed to be temporary, but the particular activity may be kept up by repeated use; the speechless man's recurring utterance is being uttered very often; it keeps up what was doubtless at first a temporary organisation.

<sup>1</sup> Of course we do not mean geographically lower, but physiologically lower, more organic, &c., and, in some instances, the centre which acts before the one called higher.

We must be very careful how we use the word "cause" with regard to disease and symptoms. We must never speak of destructive lesions causing positive symptoms. It is erroneous, I submit, to say that any sort of disease causes elaborate positive mental symptoms—illusions, hallucinations, &c.—it causes a negative mental condition, the elaborate positive mental symptoms are *permitted*. Here again we see the crudity of the expression "disease disorders the function of the brain"; destructive lesions cause loss of function of some nervous arrangements, and thereby over-function of others is permitted. Another way of putting this is to say that the effects of Dissolution are not always, never I suppose, simply those of removal of the more special, voluntary, and least and latest organised, but are very often also a permitted increased activity of the next most special, voluntary, and next least organised. Thus in every case of insanity there *are* two diametrically opposite symptomatic mental states together, and there must be correspondingly the opposite physical conditions together.

In some cases there is the utterance of "yes" or "no," or of both, without any other recurring phrase. This would be explained on the supposition that the patient was not saying, nor about to say, anything when taken ill, and thus that there were no words being revived, no proposition organising, during activity of the right side of the brain. Thus there was, so to speak, nothing to interfere with the simple course of dissolution, reduction to the most automatic of all propositions.

It is to be clearly understood that in not one of the cases do I know what the patients were saying, or about to say, at the time of their attack. I must also mention that there are observations on record which perhaps run counter to the hypothesis. Thus a man, aged 21, suddenly called out to his mother, "Oh! I feel something extraordinary inside me." These were the last words he spoke, but his recurring utterances were "No" and "Mamma." Very likely, however, calling out to his mother, he would also have cried "Mamma."<sup>1</sup>

<sup>1</sup> I take this case from a lecture by Trousseau; the utterances were in French but I give them as translated by Bazire.

I must also declare that of other cases than those expressly mentioned, I know nothing at all of particular circumstances during the onset of the ailment, which could be supposed to have given rise to speech like the recurring utterances the patients had. I have no facts to bring forward to show what were the particular circumstances during the onset of the illness of the patients whose recurring utterances were severally "man," "one," "awful," "Yes, but you know," "I'm very well," &c. At first glance it seems unlikely that a patient should say, "I'm very well," at the time she was taken ill; but as a matter of fact, people becoming ill do make such remarks. Who has not heard a person say, when a little ill, or when a severe illness was beginning, "I'm all right, let me alone," perhaps adding the contradictory statement, "I shall be better directly"?

We must bear in mind that when people are not talking they may be speaking; for there is not only external, there is also internal, speech.<sup>1</sup>

Whilst thinking on anything, at least beyond the simplest, we are using words, and when thinking on anything complex, are speaking internally. But I do not see how we are to verify the hypothesis that a patient's recurring utterance was a survival *en permanence* of the words he was internally using,

<sup>1</sup> The reader will remember that the distinction into internal and external speech (only a difference of *degree*) is not (see p. 318, Vol. I.) that made into the two *kinds* of service of words—the subjective and objective. There are degrees of each kind. The subjective, at any rate in that part of it which is "receiving speech" of others, is plainly in degrees; we hear and "receive" what people are actually saying, we also remember what they have said, which is having again, faintly, the words in ourselves which their speech had revived strongly. It must never be forgotten that when we hear and understand what any one says to us, we can only do so because our own words are revived in us; if I have not the words "gold," "is," "yellow" organised in myself, it is of no use any one saying "gold is yellow" to me. Similarly the objective is in two degrees; there is both internal and external speech, corresponding to ideation and perception. Speech in *either* of these degrees is supposed to be preceded by an automatic and subconscious reproduction of words (by a subjective proposition), just as either "internal" perception (ideation) or "external" ideation (perception) is preceded by a subconscious and automatic reproduction of images. It is not meant that, either betwixt ideation and perception, or betwixt internal and external speech there are differences only in simple degree; the degrees are, I think, triply compound.



unless perhaps the circumstances and the condition of the patient are known very exactly.

We have to consider how the hypothesis applies to the recurring utterances, No. 1 and No. 2. How is it that there is (except perhaps "yes" and "no") jargon only, or but one word only? The difficulty is greatest with regard to the jargon (No. 1), and this I shall chiefly consider. I believe the jargon to be made up of fragments of the words or phrases the patient was about to utter when taken ill.

It is, I grant, a mere guess that a speechless woman, whose recurring utterance (I spell it from the sound) was "me," "me," "pittymy," "committymy," "lor," "deah," was saying, when she was taken ill, "Pity me," "Come, pity me," "Lord," "Dear"—that her recurring jargon-utterance represented what was part of the verbal revival prior to her last propositional or interjectional utterance, that it was a corruption of syllables of some of the words which were being revived during activity in the right half of her brain, when destruction in the left half produced loss of her speech. There is a greater difficulty in supposing that such a jargon-utterance as "yabby," resembling no English word, was made up of fragments of words which were being revived during activity of the right half of the brain, when the patient was taken ill. To say that "yabby" was a jumble of syllables of some words about to be uttered will appear to be carrying the hypothesis to its logical conclusion with more determination than caution.

Let us pause to remark on occasional temporary jargon, and on something approaching to it, in other aphasic cases. Some patients, who have only defect of speech, and whose erroneous utterances are nearly always real words, may occasionally utter "jargon," as "totano." I confess that I am unable to trace the steps of the formation of such jargon. Occasionally there is a very near approach to a transposition of syllables of a word. One of my patients said "gippin" for "pigeon." There is another kind of change: one of my patients said "lamb and crobster" for "crab and lobster." There is here a fusion of the first syllable of the second word with part of that of the first, or rather, perhaps, speaking of the physical side, a result of attempts at two articulations.

Such blunders occur, I think, in persons whose speech is only very slightly defective: I mean in those who, for the most part, speak well. The following occurred in healthy people: "mukes from Boodie's," for "books from Mudie's"; "get a cash chequed," for "get a cheque cashed." A similar thing occurs in writing, as "Mear Dadam," for "Dear Madam"; "pred budding" for "bread pudding." (This is interesting, as showing that expression by writing is really speech.) I believe that these troubles of speech are owing to hurry on the right half of the brain, to hurried reproduction of the words of the subject-proposition. Believing that images and words are subjectively revived in an order the reverse of that in which images and words are finally arranged, I suppose, speaking roughly, that the words of the subject-proposition "come over" to the left side prematurely. These utterances are jargon, the sources of which we can trace, in so far, I mean, as fusion of an initial syllable of a second word with that of a first word goes. And I believe this to result from hurried action in patients who have defect of speech, partly for the reason that such and similar mistakes occur when a patient replies at once to questions abruptly put to him, and because when not hurried, when not "taken suddenly," he may speak for the most part well.

We can now return to consider more easily the permanent recurring jargon of patients who are speechless. We speak of what is supposed to have occurred in the right half of the brain, when the speechless patient was uttering, or about to utter, his last proposition. If the reader does not go with me in supposing that the right half of the brain is concerned, he must, at any rate, believe it is some part other than that on the left which the disease destroyed. It is suggested that when the recurring utterance is in clear propositional form, such as "Come on to me," the right half of the brain was acting normally or usually; that when it is a single word or jargon, it was acting abnormally, being "hurried." What is the cause of this hurry? I believe it to be strong emotion. At the time of the onset of the illness, we may suppose strong emotion would be induced by the external circumstances, or much more probably by the setting in of the

illness, the patient being afraid of worse to come. Here, of course, a knowledge of the patient's temperament is very important. The patient who uttered "me," "me," "pittymy," "committymy," "lor," "deah," was, to my own observation, a most excitable woman. Her husband used to remark on this. He said she was fond of sensation tales: she would any time go out in the night to a fire. Strong, especially suddenly induced, emotion implies, on the physical side, great nervous excitation, that is to say, strong, wide, and sudden discharges; there will, under strong emotion, be more rapid and more numerous, and therefore conflicting, discharges of nervous centres. In the cases under remark we consider the result of discharges on centres for words.

The result of numerous sudden, strong, and therefore conflicting discharges, would of course be the survival of the fittest, but the survival amidst too numerous and too strong discharges would not be an elaborate and perfect proposition. We suggest that it would be but one word or a jumble of syllables of some words. Of course "fittest" here does not mean "the best," nor the fittest *for* the *external* circumstances of the time; it is the survival of the fittest under the *internal* circumstances.<sup>1</sup> The fittest words or syllables are the victorious

<sup>1</sup> The words and images which survive during healthy discharges are survivals of the fittest. So it is in disease. The images that survive in delirium are the then fittest, considering that some of the highest nervous arrangements are *hors de combat*. The delirious patient is not the same person as that patient before his delirium set in; the delirious patient is the same man *minus* more or fewer of his highest nervous arrangements; his delirium is the fittest mentation then possible. In all cases it is supposed that what becomes conscious is what survives at the end of a conflict; the conflict is almost at zero in highly organised processes, such as those for recognising very simple and very often seen things; in these cases consciousness is almost at zero too. In movement of any part of the body there is not only co-operation, but antagonism also of muscles or muscular strands (Duchenne uses the expression "co-operation of antagonism"). The antagonist pulls—those against the direction of the displacement to be effected—may be symbolised as minus quantities; the pulls co-operating in that direction as plus quantities. A movement is then not the arithmetical sum of pulls of all the muscles engaged, but the algebraical sum of the pulls. Transferring these ideas to antagonistic and co-operating discharges of nervous centres, we may suppose that what we have called, both for health and disease, the survival of the fittest during a conflict of numerous nervous discharges is the algebraical sum of what on the physical side are co-operating and neutralising discharges during the conflict.

words or syllables which have survived during a conflict of very strong and sudden discharges beginning deeply in the nervous system, and ending, so far as we are now concerned, in that of many nervous arrangements for many words. Such survivals may be little fit or not fit at all to the then external circumstances; they express the emotions and ideas induced well or badly, as well as it can be done under the circumstances.

It is supposed that jargon is a survival of some of the syllables or articulations more or less fused during the sudden termination or arrest of a strong conflict of discharges of numerous nervous arrangements for different words. It is a popular doctrine that strong emotion leads to incoherent utterances, which, I suppose, means the survival of words symbolising more than one different set of ideas. Strong and suddenly induced emotion may lead to temporary speechlessness. We suppose that temporary speechlessness with great emotion betokens very numerous and strong nervous discharges, conflicting so as to balance or neutralise one another.<sup>1</sup> In some other cases of disease psychical states cease during sudden excessive discharges. An illustration is that during sudden and strong discharges, epileptic discharges, beginning in the very highest nervous arrangements (consciousness), the substrata of consciousness cease.

We now consider the recurring utterance of one word (No. 2, Vol. II. p. 205). Let us first note what occurs in health. Very often under strong and sudden emotion, a normal or usual degree of speech is not possible; some inferior speech is possible. When there is not incoherence, but one word may be uttered or rather exclaimed, as "fire!" "help!" the utterance is indeed often a very simple interjection, as "oh." One word, as "fire!" might in normal circumstances be propositional only or so practically; it might *mean* "there is a fire," but exclaimed under strong emotion it is also largely of interjectional value. Strong emotion leads to inferior speech, to

<sup>1</sup> Probably there is a more complex condition; during strong emotion, there are rapid and strong discharges on the vital organs which soon lead to exhaustion of these organs; thus there will be a multiplication of effects; the cerebrum, especially its highest, centres, will be ill served with blood.

more automatic, more organised utterance. Figuratively speaking, emotion uses propositions in a largely interjectional manner, that is to say, it reduces them to or towards inferior speech. Emotion, as it were, still speaking figuratively, appropriates and subordinates an intellectual utterance. There is at any rate an interjection in the making, in the exclamation "fire!" "help!" &c., a degradation of speech (compare swearing). We see in the utterance itself, that with a fall in the intellectual element there is a rise in the emotional one. There may be an equal, no doubt a greater, liberation of energy during these utterances, but it is directed more strongly on the vocal (including respiratory) organs, to those organs which serve especially during emotions. Strong emotion tends to more automatic, inferior, utterance.

I suppose then that strong emotion, or rather, of course, the nervous discharges accompanying it, may have led in the becoming speechless man to so rapid a discharge of so many nervous arrangements of higher cerebral centres in this case for words, that in the great struggle resulting, the survival of the fittest was a survival, not of the best for the then external circumstances, but of but one word, or of some syllable or syllables; the fittest was then an interjection, or only some parts of sentences or parts of words fused into jargon.

Rapidity of emotion is the great thing in the above considerations. In another way we are concerned with rapidity—with rapidity of the being taken ill, which means rapidity of destruction of nervous arrangements in the left half of the brain.

In all cases of nervous disease we must endeavour to estimate most carefully the element of rapidity of lesions, not only the quantity of nervous elements destroyed, but the rapidity of their destruction. We have to try to estimate the Momentum of Lesions. We however use the term gravity of lesions. I do not pretend that I can show the bearing of the factor rapidity on symptomatic conditions in cases of Affections of Speech. It is fair, however, to consider it in this regard to suggest a basis for future investigation. The slowness with which a lesion comes on is determined by the slow onset of the symptomatic conditions; a small sudden hæmorrhage produces

greater but a more temporary effect, provided it does not at once kill, than a large, slowly developed softening. But in the latter the paralysis or other defect depending on mere destruction of the centre is more local and more lasting.

A very sudden grave lesion would render unavailing all external circumstances, and would prevent all emotion, for it would produce unconsciousness at once; a less grave one would prevent the influence of external circumstances, but would develop emotion. A lesion of very little gravity would allow external circumstances to act, but to act quietly and allow clear propositions to be framed. Repeating in effect what has been said, it does not follow that the lesion in the latter case is not an extensive one; the question we are now about is as to the gravity of lesions, metaphorically speaking *mv* of lesions.

We should then note whether the aphasia and hemiplegia are rapidly produced, whether or not there be sudden loss of consciousness, and note also the length of the coma, if there be any. Everybody does this for clinical purposes, in order to obtain empirical evidence of the kind of pathological process, whether it be clot softening, &c.; but we should use the facts also as regards the interpretation of the kind of recurring utterance the patient may have. Perhaps the bearing of rapidity of destruction on the nature of recurring utterances is not made evident in the above. It is not here *directly* a question of rapidity of destruction of any centre, or of "shocks" given to centres connected by fibres with the one suddenly damaged, but a question of the indirect effect of rapidity of destruction, of such destruction in so far as it is a *sudden removal of control* (or inhibition) over lower centres. Of course by "lower centre" we do not mean one geographically lower, but one anatomically and physiologically lower. The destruction can only directly *cause* negative symptoms; it neither directly causes the utterance nor interferes with it; it may be popularly said to cause it and to affect it indirectly, but it is better to say it permits it. In so far as cases of aphasia are concerned, the above is hypothetical. Let us consider the principle stated more generally. In other cases of nervous disease rapidity of dissolution is, I

think, evidently an important factor with regard to symptomatic conditions. The epileptic maniac is the most furious of all maniacs. Why most *furious*? The fury is the psychical side of what is physically of course great activity of nervous arrangements; it is activity of lower nervous arrangements; for as the furious maniac is insensible also, it is plain that his highest nervous arrangements are *hors de combat*. Why then are the lower nervous arrangements so active? I believe it to be because the process of dissolution effected in the epileptic paroxysm prior to the mania is the most rapid of all processes of dissolution—control is most rapidly removed. Let me try again to make this clear by formulating it otherwise. We have not only to consider “depth” of dissolution, but the rapidity with which the dissolution is effected—control removed. I believe we may say that the deeper the dissolution, the more general and more automatic are the processes remaining, and that the more rapid the dissolution, the more excitable are the nervous arrangements for those processes. So that with shallow and yet rapid dissolution the actions are elaborate and also busy; with deep and rapid dissolution, they are simple and also violent (it is not a question of “importance” of actions).

I presume then that the more rapid the destruction of the part in the left half of the brain, the more excitable would be the nervous arrangements in the corresponding undamaged right half, and thus that the activity of it would be greater—greater for some time after the onset of the illness. But the more rapid the destruction, the more quickly there would be loss of consciousness; thus the then external circumstance would be unable to act at all, and although the lower centre might be more excitable than usual, there would be nothing for it to manifest as a result of what was saying or doing at the onset of the illness.

To recapitulate. By considering (1) the external circumstances at the time of the being taken ill; (2) the intensity of the emotional state under which the last attempt at speech was made; and (3) the gravity of the lesion, we may perhaps be able to show why this or that kind of recurring utterance remains in particular cases of speechlessness.

We shall now consider phenomena analogous to recurring utterances in some cases of defect of speech (No. 1, Vol. I. p. 314), in order to widen the basis of investigation.

The patient who has defect of speech may get out a word, right or wrong, and go on uttering it; or he may even get out a proper reply, such as "very well," and go on uttering that in rejoinder to further questions to which it is irrelevant, being aware of that irrelevance. To use Gardiner's expression, who drew attention to this peculiarity, the patient gets the word or phrase "on his barrel-organ." It becomes a temporary recurring utterance; the permanent recurring utterance in loss of speech may be called permanent barrel-organism. In cases of defect of speech the patient may write a little, and then go on writing the same syllable or word or phrase over and over again. Here again is evidence, although indirect, of "barrel-organism" of words or syllables.

The following is a letter written by a patient who had defect of speech: it was not punctuated. He first wrote his address correctly, which for obvious reasons I do not give, I substitute "Hurst Row, North Newington Road" for it; the address recurs in the letter; the factitious address is put in place of it:—

"Dear Sir. I feel very well just now" ("for now" and "I feel" came next, but are crossed out) "for Hurst Row I feel very well just now for thingg in the first way for the thamk now. I know now I was in the first now in the first now in the Newington Road I keep you first way in Newington Road the poor way is the best way is the best way for me is the best way for me is the best way for me is the best way for me is the best way for me is the best way for me for ways kept for me for kept ways kept me for way kept for me."

Here ends the letter. There is, however, plainly something more than barrel-organism in it.

From what would be generally called the physical sphere we obtain illustrations in some cases of *loss* of speech of "barrel-organism." If, after getting an aphasic to protrude his tongue, we ask him to put his hand on his head, he may, instead, open the mouth and perhaps put out the tongue. This, I grant, may be of doubtful interpretation.



Here let us note another peculiarity in cases of defect of speech, which, perhaps, can only be shown to concern us indirectly in regard to what has been said. It is a very important matter in the elucidation of the nature of defects of speech, and may properly be mentioned now in order to suggest inquiry if there be anything analogous in the recurring utterance in cases of loss of speech. In some cases of defect of speech we find that the patient who has uttered a word or phrase correctly, and who goes on re-uttering it as a reply, cannot repeat it when he is told and when he tries. He has said correctly, "I don't know." We tell him to say "I don't know;" he fails. Whether there is anything strictly analogous to this in the case of permanent barrel-organism Nos. 1, 2, and 3, as there is of "no," No. 4, will not be easy to determine. For if we ask the patient to *say* his elaborate recurring utterance, just as we ask patients who reply "no" to say that word, there is the difficulty that the recurring utterance comes out when they *try* to say anything; and thus its coming out may be not saying it, but simply uttering it as at any other time. I confess I have no personal observation on this matter worth mentioning. It has long been known that some aphasics at least cannot say parts of their recurring utterances. Thus a patient of Bazire's uttered "sapon, sapon," but could never say "sap" or "pon" only. Trousseau makes analogous observations. But here obviously there is a difficulty. What we call part of a word is really another word or syllable. A patient who consulted me for loss of speech learned to *say*—not merely to utter—"Battersea"; but she could not say "batter." When asked to say it, she said "Battersea."

Here we may make further remarks on dual mental action, taking this time not the process ending in ideation or perception, or both, but the process ending in voluntary, as distinguished from automatic actions. What we now say may serve to integrate remarks which necessarily came now and then incidentally in discussing different parts of our main topic. We make a few preliminary observations.

We must bear in mind that "will," "memory," and "emotion" are only the names men have invented for different

aspects of the ever present and yet always changing latest and highest mental states, which in this locality constitute what we call consciousness—consciousness being really a name for a series of varying and different consciousnesses. There is, however, a double series; subjective and regular consciousnesses. Each one of the series of object consciousness is secondary to a state of consciousness or subconsciousness, representing us altogether subject-consciousness. It is this duplication, I suppose, which gives us the feeling that we have a sort of general and persisting consciousness, and are also now, now, and now, conscious, this or that particular way, and which makes us say that this or that sensation or image comes *into* consciousness; or that we have a sensation and know that we have it.

Taking “will,” “memory,” and “emotion” to be real independent entities or faculties reminds one of the old woman’s remark, “How lucky it was that Adam called all the animals by their right names.”

To say a word or proposition when told, “for the sake of saying it,” is not a language process at all. A speechless man’s inability to say “no” when told (see Vol. II. p. 213) is a thing of the same order as his inability to protrude the tongue when told. The consideration of such facts will help us to classify the phenomena of cases of aphasia on a deeper basis than that of language. To use an expression somewhat loose in this connection, there is loss of certain voluntary actions in some cases of aphasia, with conservation of the more automatic—a dissolution affecting more than language processes, and affecting language processes not so much as language processes, but as they are some of the voluntary actions. We have to consider speech on this wider basis in order that we may be better able to see how speech is part of mind, and thus to get rid of the feeling that there is an abrupt and constant separation into mind *and* speech.

It is in considering the nature of these phenomena that we see, I think, the bearing of Spencer’s remark on the distinction betwixt voluntary and involuntary operations. There we have to do with duality of mental processes. In the voluntary operation there is preconception; the operation is nascently

done before it is actually done ; there is a "dream" of an operation as formerly doing before the operation ; there is dual action. Before I put out my arm voluntarily I must have a "dream" of the hand as being already put out. So too, before I can *think* of now putting it out I must have a like "dream," for the difference betwixt thinking of now doing and now actually doing is, like the difference betwixt internal speech and external speech, only one of degree ; in one there is slight discharge of a certain series of nervous arrangements, in the other strong discharge of that series. The "dream" must occur before I either think of now doing something, or before I actually do that thing, just as words must be reproduced in me before either I can say them to myself or aloud. To say that we know what we are going to do, or that we are intending to do this or that amounts to admitting the above hypothesis. These expressions imply our having a nascent excitation of nervous arrangements representing the parts formerly concerned in doing that which is now to be done again. If we say we are trying to do something, we mean similarly, and so we do if we say we remember how to do anything ; there being no "faculty" of remembering an action apart from having the action (as it was formerly doing), again actual, although faintly actual, nascent. To "will," to "know," to "intend," to "try," to "remember," have each, in their several contexts above, the same meaning. They are different names for the subjective reproduction which precedes objective reproduction ; they are names for "dreams" of what was once doing, and is now to be attempted again. Indeed the simple expressions, "He speaks," "He does so and so," imply the duality ; imply objective activity following subjective activity. "He speaks," or "He does," implies a then temporary duplication of a person into subjective and objective ; "he" represents that person's whole self of course, and "speaks" or "does," represents something doing by his whole self. "He" represents the whole of him in the stage we have figuratively called having a "dream" of a past operation, "speaks" or "does" represents the whole of him in the stage of imitating it. (We say figuratively, because we do not mean a visual dream, but having again sensations representing parts of the body dis-

placed, and as being moved thus or thus.) To take a simpler case, in speaking of which, however, usage will not allow application of the terms "voluntary" and "involuntary." "He has this sensation." Subjectively, there is a sensation arisen in him which associated directly with others, and by them indirectly with all others—all that have been organised. Objectively there is disassociation, the sensation is considered separately from all others that are organised and in relation to those now organising. We may fancifully put the duplication thus: "*He* has it," and "He has *it*;" thus artificially separating what is a rhythm of a subjective and objective state. When then we assert that a speechless man tries to say "no" and fails, we are tacitly affirming that he has the word "no" revived in himself, a "dream" of the word. He has the subjective word "no," but not the objective word "no," there being really two "noes," all words being in the duplicate of subjective and objective. So, were we to use popular language, and to say of a patient who "tries" to put out his tongue, and fails, or who tries to say "no," and fails, that he has lost part of his volition, we should only mean that he had lost not a part of some faculty, but the very objective actions themselves, their nervous arrangements being broken up; he has lost a part of himself. And were we to say that the patient desires to do those things, we should only mean that he retains the subjective actions, which in health are precedent, and are to him the preconception of the objective actions which cannot follow, for the simple reason that they do not exist. I suppose such expressions of duplication of mental states, "*ideas of actions*," "*memory of words*," "*feeling, a sensation*," and "*knowing that we have this or that feeling*," &c., imply that there is first and subconsciously the action, or the word, or the particular sensation associated with (because arisen out of), the already organised in our whole selves (subjective actions, words, &c.), before the second reproduction of them or rather of their duplicates in relation to what is then organising from the environment. A subjective word is first a centre of association with all other organised words, symbolising all other images, and afterwards a symbol of a particular image; an objective word is first a symbol of a thing, and next acquires

new associations with other words, then using along with it and similarly arisen to indicate new relations of things in the environment.

In healthy speech it is hard to see how we can have a proposition (either in the degree of internal or in that of external speech) before the words of that proposition have been subjectively revived. Otherwise how should we be more than machines? Words must have arisen in us in an order symbolising states arisen in us, before a proposition we utter can have *for us* any meaning as symbolising external relations of things one to another in the environment. To use popular language, we must first remember the words and then say them.

We do not, as already incidentally implied, mean that the parts of the subjective and of the objective process are arranged in the same order.

Let us return to the simple movement of the arm. The "dream" here begins of course with that which corresponds to what is the end of some operation which was actually done on some former occasion. If I am to put out my arm so far I must have a "dream," *beginning with* the hand being so far put out; and next of it as in varying positions up to that state of rest from which the movement really began when it was formerly done. If this were not so, we should not know what we were going to do; the end must come first subjectively, before the objective process can in any voluntary action begin, and can go on to that end. This implies that two different nervous units are engaged. One of the twins represents (*a*) incoming currents from skin stretched, muscles moving or balanced by antagonism from joints, &c., in the limb engaged, and (*b*) currents coming from the rest of the body, as being thus or thus fixed, or as thus or thus variously displaced during the particular local movement of the limb. And of course there is an accompanying series of outgoing currents, for if there were no motor element we should have no knowledge of the relations of the parts moved to one another and to the rest of the body; incoming currents would not suffice to give this relation any more than incoming currents from retired elements, without accompanying ocular movement, would

suffice to give us a knowledge of the relations of these elements one to another; and, plainly, unless retired elements are first known as out of one another, impression on them by objects would give us no notion of the exterior of those objects. Similarly, until we have already a knowledge of the relation of parts of the body one to another, we could not begin to make any movement of part of the body to operate on the environment. During discharge of this unit a psychical state of what was formerly doing arises. When the operation is to be repeated, to be done now voluntarily, the other unit discharges the operation, starting from an attitude of the body and passing on to the end of the operation by the limb. Manifestly this must begin by an attitude of the body, and the progress in the arm will be from the shoulder to the hand, in an opposite order to the "dream-movement." It is during the play of these two movements that what is called muscular sense arises. The psychical side of one process is part of what we call desire, of the other, of what we call effort.

That subjective states arise in an order the opposite of that they are arranged in in corresponding objective states is supported by the analogy of some dreams. A noise develops a dream, but, sometimes at least, the noise, which in reality acts on the sleeper first, is last in the dream it excites. In all dreams provoked by local excitations I suppose the transfigured excitation comes either last or later than the excitation itself. And I suppose that in such a proposition as "gold is yellow," the subjective order is the reverse of the sequent objective order; for our concern is first with the yellowness of gold, not with gold. This view is, I think, in harmony with what was said on transposition of syllables in mistakes of the slightly aphasic and the healthy. I believe there to be duality also in automatic operations; but in this case the movement has so often followed the dream that the two are nearly equally perfect and easy. The voluntary operation, the prior "dream," is imperfect, or, if perfect, the movement done after it does not imitate it closely; we try to do so, and fail. Becoming automatic by repetition is, on the physical side, for the two units to discharge more nearly together, because lines of less resistance are established; there is less delay betwixt

them, and thus preconception through the discharge of the first unit and less sense of effort during discharge of the second. Thus less and less consciousness attends processes the more they become automatic; subjective and objective actions become, as it were, nearly fused.

Let us now take other kinds of nervous affection which show something analogous to permanent barrel-organism of speechless patients. We wish to point out that there is evidence that operations going on at the time of unconsciousness supervening remain nascent or in abeyance—are not always swept away—during the unconsciousness. During the restoration to consciousness they become active again; on full restoration to consciousness, they cease.

Directly after or in coma from various causes, we occasionally see a reversion to actions doing when the comatising lesion occurred. An ostler coming round from coma, due to the kick of a horse he was grooming, began to “hiss,” as grooms do when engaged in rubbing down horses (Brodie).<sup>1</sup>

Abercrombie writes: A lady, whose case has been communicated to me, was seized with an apoplectic attack while engaged at cards. The seizure took place on a Thursday evening, she lay in a state of stupor on Friday and Saturday, and recovered her consciousness rather suddenly on Sunday. The first words she then uttered were by asking, “What is trumps?”

I suppose the above instances show that actions nascent at the time when the illness occurred remained so during the coma, and went on again actively when consciousness was being restored; on full restoration to consciousness they ceased; the barrel-organism was temporary, because the highest nervous centres were only temporarily *hors de combat*.

The following case, kindly supplied to me by Dr. Buzzard,

<sup>1</sup> I suppose the following to be analogous, although consciousness was only partly restored: the action of laying the oilcloth had become “barrel-organic.” A woman I was asked to see in a surgical ward of the London Hospital, who fractured her skull by a fall when laying down the oilcloth on a staircase, kept, during partial unconsciousness a few hours before death, manipulating the counterpane of her bed. We supposed this to be a continuance of the action of laying the oilcloth; the patient desisted for a time, at least when the nurse assured her “it was properly laid.”

has never been published. It may be taken as a faithful record, since the observer's qualifications as a neuropathologist are of the very highest. It is one of extreme value in many ways.

"About fifteen years ago, a medical friend of mine, some 57 years of age, was pitched on to his head, owing to the horse which he was riding having put his foot in a drain. He was taken home in a state of profound insensibility, in which I found him very shortly afterwards. From the symptoms it was probable that he had fractured the base of his skull. For some hours (I have no note of the number) he lay in a state of deep coma, incapable of being aroused. From this his condition gradually changed to a state in which he appeared to sleep, but would reply 'Yes' or 'No' to questions put to him. He did not appear to notice anything about him, and never volunteered a remark, but scarcely ever failed to reply in the way described, and with such an amount of propriety that I remember it was difficult at first to believe that he was unconscious. Offered food, he would sit up in bed, drink it without a word, lie down again and go to sleep. On one occasion I wanted him to take a common black draught, but he appeared to resent this, said 'No! no!' fiercely, and turned away. By a little persistence, however, he was induced to swallow it. He would get out of bed, take the chamber utensil out of its place of concealment, and pass urine regardless of any one in the room.

"One morning, to my astonishment, he got up, walked to his shaving-glass at the window, and proceeded to lather his chin. He then shaved himself, with very fair completeness, and returned to bed. I happen to know that this was the way in which he invariably commenced his morning toilette, for he was a late riser, and he had frequently risen and shaved himself during my visit. From this day he got up every morning about the same time, repeated the operation of shaving, and then returned to bed. The action was performed with the same evident unconsciousness of the presence of others which we see in the somnambulist. Having returned to bed he would lie for the rest of the day in a dozing state, which was only interrupted by his taking food and passing his



evacuations, which actions were evidently performed in a purely automatic manner.

"Upwards of a fortnight from the time of his accident he woke up into consciousness, noticed persons in the room, and made some slight remark. Little by little during the next hour or two he began to converse at greater length, referred to circumstances relating to his business which had occurred before his injury, and it became presently evident that the time during which he had been in bed had left no trace upon his memory. Of the injury itself he knew nothing whatever, the last thing which he remembered being that he was riding on horseback.

"When asked the day of the week and month he unhesitatingly gave the date upon which (more than a fortnight previously) he had met with the injury, and became quite angry when assured that he was mistaken. The period of his illness had been cut out, as it were, of his life in the cleanest and most complete manner, and not a trace of it has ever since presented itself to his mind."

In the above-mentioned cases the patients recovered, and thus their cases are not closely analogous to our cases of permanent barrel-organ utterances in aphasia. A patient who recovers soon from aphasia loses his recurring utterance, becomes able not to utter it. These cases are analogous, and so is the temporary barrel-organism of some patients with defect of speech. In the permanent barrel-organism, or recurring utterances of patients who remain speechless, the restoration of the part (in the left half of the brain) placed *hors de combat* is impossible; it was destroyed altogether. The following case is more closely analogous to these cases than the above. Abercrombie relates (after Conolly): "A young clergyman, when on the point of being married, suffered an injury of the head by which his understanding was entirely and permanently deranged. He lived in this condition till the age of eighty, and to the last talked of nothing but his approaching wedding, and expressed impatience of the arrival of the happy day."

What does recovery in the case of the cures of barrel-organ actions mentioned mean? It means restoration to

functions of some or enough of the highest nervous arrangements; it means return of control. But in cases of speechlessness from entire destruction of nervous elements there can be no restoration; the control does not return. The recurring utterance remains, and by repetition becomes highly organised.

# THE THERAPEUTIC VALUE OF CITRATE OF CAFFEIN AS A GENERAL SEDATIVE, ANODYNE, AND DIURETIC.

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CITRATE OF CAFFEIN, which exists in the form of silky prisms soluble in about sixty parts of water, has hitherto obtained a certain amount of recognition as an anodyne in varied cases of neuralgia. In hemicrania, tic-douloureux, dorso-intercostal neuralgia, and in some spasmodic affections such as asthma, it has occasionally been found of service; but beyond a passing admission of stimulating properties, its use has been for the most part empirical, and the drug, whether from the want of appreciation of the pathological causes surrounding the disease, or from the insufficiency of the dose usually prescribed, has been very generally regarded as of too doubtful efficacy for anything approaching universal recognition.

Elsewhere ('The Practitioner,' Jan. 1879) I have recorded my observations on the use of citrate of caffein as a diuretic in cardiac dropsy, and in doing so endeavoured to point out from a few selected cases the reasons which led me to its successful employment in a *class* of cases simulating one another in certain well-recognised pathological characteristics. Subsequent investigations have confirmed these observations in such an unmistakable manner that I may be pardoned the desire to enforce my conclusions, and at the same time to direct attention to the general therapeutic position of citrate of caffein as a sedative and anodyne in those conditions of mental anxiety and exhaustion, melancholia, sleeplessness,

morbid distress, asthenic mania, and neuralgia, where, as so frequently occurs, a similar train of pathological causes will be found (only developed in a slighter degree) as when a heart failing through pronounced adynamy or defective compensatory power, becomes associated with states, well defined, of arterial relaxation and passive venous engorgement.

In all cases of advanced cardiac adynamy with the physical signs of an embarrassed inco-ordinate cardiac muscle weakened from dilatation and overwork, there will in all probability be the associated condition of defective nerve nutrition. The restlessness and nervous anxiety, the chronometric derangement between respiration and cardiac impulse, the distressing wakefulness yet eager longing for repose only to be met by a turbulent cycle of ever-recurring thought, are indications of a brain morbidly nourished and lacking repose. The small flaccid pulse again, the want of harmony between the cardiac and arterial pulsations, the spasmodic urgent dyspncea, and the localised or general anasarca may be the mechanical consequences of a heart dilated and wanting power, of an arterial system unbalanced, and of a venous system overloaded; but may they not also be the indications of a central and peripheral nervous system enfeebled and impoverished as well as venously congested from circulatory disorder? Vaso-motor paresis and diminution of blood-pressure will be admitted as prime factors in the train of symptoms enumerated, and with stimulation therefore of the cardiac ganglia, and stimulation of the motor nerves of the peripheral arterioles, we may do much in assisting to restore that equilibrium in the circulation which shall promote arterial fulness, remove effete material by the proper channels, and moreover afford such a fresh supply of nutrient blood to the heart, brain, and system as shall induce sleep and vigour of mind, the consequences of normal nutrition.

In advanced cases of cardiac disorder then where muscular embarrassment and neurol inco-ordinate cardiac action, the indicators of progressive mural decay, have with their consequences to be met, citrate of caffein in doses of from gr. iij. to gr. v. may be looked to (1) to induce co-ordinate action of the heart, (2) to increase vascular pressure and

promote free and almost instantaneous diuresis, and (3) to stimulate a heart gradually being relieved of its source of embarrassment to more vigorous and forcible contraction. By these several means citrate of caffein is an important *aid* to the tonic action of digitalis; for without it, and unless the heart and circulation is relieved (either by diuretics establishing the necessary drain on the general circulation and indirectly staying the congestion of the renal veins in their connection with the inferior vena cava, or by drastic purgatives acting on the valveless portal circulation), tonic doses of digitalis will only make a further demand upon the embarrassed muscle to contract more forcibly on its overloaded chambers. With caffein however, on the other hand, as a diuretic of considerable power we hope to establish an outlet from the general system which, coincidently with more powerful cardiac contraction, shall relieve the stagnating pressure of venous blood on the right side of the heart; and by promoting equilibrium in the cardiac and general circulations, shall pave the way for the use of cardiac "tonics" as safe and efficient or even curative remedies.

For an explanation of the action of citrate of caffein upon the heart and vessels we must look mainly to the sympathetic system. The intrinsic cardiac ganglia endow the heart with that inherent power which enables us to account for the occurrence of palpitation of the heart notwithstanding the existence of adynamy and arterial relaxation, and notwithstanding the clinical fact that in many instances the pulse is no reliable indicator of the force, rapidity, power, or character of the cardiac action.

In the sympathetic system special to the heart itself, and in the cardiac plexus of the great sympathetic with its connecting media to the spinal ganglia and cord (the cervical sympathetic and cardiac nerves), we find, it must be remembered, a depressing as well as an accelerating power and not an accelerating power alone. The heart exhibits in itself the excitory ganglia (specially of Remak and Bidder) counterbalanced yet naturally uncontrolled by the inhibitory interauricular ganglion of Ludwig, and all these may severally be stimulated by sensory centripetal fibres taking origin in

the endocardium. Thus the heart has an inherent or dissociated power; and notwithstanding the existence of arterial relaxation there may nevertheless be palpitation adynamic and inco-ordinate, the result of reflex irritation commencing either in the viscera or also in the heart itself. The relation of the vascular system to the heart through the agency of the nervous system is of no less importance; the cord has an indirect influence upon the heart as acting upon the peripheral circulation, and according to the observations of Ludwig this influence is principally exercised upon the vascular supply of the abdominal viscera through the medium of the splanchnic nerve. But Cyon has still further shown that the change in the peripheral circulation, the result of a vaso-motor modification, may be a reflex phenomenon caused by excitation of a sensory nerve beginning in the heart. The "depressing nerve of the circulation" of Cyon represents in other words the centripetal origin of a reflex action, producing diminution of blood-pressure in general from paresis of the vaso-motor nerves, and depletion of the heart. The train of pathological conditions which we have been briefly considering may therefore be the result, or at least partially dependent upon, some irritating effect acting upon the substance of the heart itself. Clinical investigation will often speak of a heart dilated, adynamic, embarrassed, failing from useless work, with right side engorged with venous blood; and therapeutically we shall desire to stimulate the cardiac ganglia reflexly excited through pressure of blood on the endocardium to co-ordinate action, and, at the same time, whilst relieving a stagnating venous circulation, to increase vascular pressure, and to promote cardiac contraction through the medium of the peripheral and especially the splanchnic nerves. These are the effects which we have reason to think are induced in advanced cases of cardiac dropsy from progressive mural decay when the advent of free diuresis and a co-ordinate and more forcible cardiac action are amongst the clinical indications of the establishment of a more healthy circulation.

But there is something more resulting from the action of citrate of caffein in these cases of cardiac distress and dropsy beyond the mere establishment of diuresis and the stimulation

of the heart muscle. A patient in the severest distress, wakeful and disturbed with urgent dyspnœa and throbbing vessels, appears to pass the first quiet night with refreshing sleep under the influence of caffein. In the condition of insomnia associated with non-compensated valvular disease and progressive dilatation, there are it must be observed two conditions to be met by remedies for the production of sleep: (1) a disorder of the cerebral vascular system, resulting in arterial relaxation and venous congestion; and (2) a state of malnutrition or impoverishment of the cerebral cells, resulting in a morbid state of their activity. The venous engorgement is passive, and is a consequence of the atonic or obstructive lesion of the heart forbidding or preventing the onward flow of blood; but the state of arterial cerebral relaxation<sup>1</sup> (so often described as "dilatation," of which there is no clinical or pathological evidence) is, moreover, the result of a vaso-motor paresis of the intracranial blood-vessels, which more than probably invariably results when an overtaxed brain, approaching exhaustion, becomes in its clinical manifestations morbidly troubled and sensitive, and even in the later stages betokening the end—delusive, irritative, and full of wandering, illusory thoughts. The irritation and malnutrition of the cerebral cells in the second place is mainly dependent upon the want of elimination of nitrogenised waste, an inevitable result of the scanty secretion of urine and want of interchange of elements, and the form of sleeplessness closely simulates in its clinical manifestation that due to lithiasis; there is the same anxious desire for repose, but the cerebral activity forbids anything but a barren and irksome cycle of thought; there is no supply of fresh nutrient material, and the venous engorgement and state of arterial relaxation forbids the elimination of effete material. The free diuresis, therefore, which citrate of caffein insures, must be looked upon as one of the most important factors in the train of processes which it aids in establishing. The

<sup>1</sup> Niemeyer supports this view, for in speaking of cerebral hyperæmia he is careful to point out that "in non-compensated valvular disease of the left ventricle the whole amount of blood in the vessels of the brain and its membranes is not increased, since while the veins are overfilled the arteries are less full"; and he adds that capillary hyperæmia only results from "the overflowing of the veins obstructing the flow of blood from the capillaries."

brain partakes with the other organs of the body in recovering its normal state of vascularity and vascular equilibrium; effete matter is eliminated; and with the approach of the proper state of nutrition of the cerebral cells we may hope for the establishment of that normal physiological state of cerebral anæmia upon which refreshing sleep depends, and which cannot be attained so long as a state of vaso-motor paresis prevents anæmia as much as it prevents normal circulation and fulness of vessel.

These views as to the action of citrate of caffein in cases of cardiac dropsy of a certain type, lead almost imperceptibly to the explanation of the remaining cases in which caffein may be looked to to afford relief. That coffee in the form of strong decoction should have been recognised in medicine as an astringent in certain forms of diarrhœa is scarcely to be wondered at: it probably acts on the theory of stimulating the vaso-motor nerves, and so relieving the atony and consequent relaxation of the mucous membrane, allaying such forms of diarrhœa as may be deemed atonic, colliquative, sympathetic, or vaso-paralytic, as in cases of extreme cachexia. In asthma pure *café noir* has the recommendation of Dr. Hyde Salter; but according to the recent remarks of Dr. Thorowgood ('Lancet,' 1879), who speaks encouragingly of the use of citrate of caffein in doses of from one to four grains in the same disease, "its chief use is probably in protracted cases, where the nervous system appears to be worn out by the protracted state of spasm." In migraine, again, the citrate of caffein has been used with a certain degree of success. Like guarana, it may be looked to in some cases to ward off an attack if given sufficiently early; or in the later stages, when the nervous system is becoming exhausted, a patient who is a constant sufferer will often be able to tell the stage when a dose of a stimulant such as caffein will obliterate the lingering traces of the attack, and perhaps induce sleep. There is, in addition to this clinical evidence of the value of caffein, the observation of M. Hervey, that migraine is an arterial neurosis taking origin in the great sympathetic nerves, and its seat in the nervous filaments accompanying the arteries, the result being dilatation (or perhaps relaxation) of vessels. Collateral evidence indeed appears



to show that in a *class* of diseases associated with or dependent upon a vascular neurosis, with relaxation of vascular walls, citrate of caffein may be looked upon as remedial. It is only an anodyne, and its lasting effects as a sedative can only be expected, if associated with the "nerve storm" there is arterial relaxation and diminished cardiac energy as the prominent indications to be met.

With such pathological elements, we have a vast and important class of diseases in which we may obtain important results. In melancholia, in the brain of overworkers, in the sleeplessness and depression of spirits of drunkards, in poisoning by opium or aconite, and in such forms of acute mania as have been called by M. Gubler "asthenic," where a defect of incitation, an eye pale, and a pupil large, indicate excitants—in all these and allied cases we may reasonably hope that citrate of caffein, so readily given hypodermically, will find a permanent place. As regards the administration of the drug, it should be given in sufficient doses. Three grains of the citrate should be looked upon as the minimum dose; in cases associated with pain or asthenic mental excitement the dose will probably have to be increased and be frequently repeated; but in cases of heart disease, where it will serve as an adjuvant to digitalis, three to five grains given every night will usually be found sufficient to establish free diuresis, and induce that proper state of nutrition from which sleep, as not the least of the important ends to be attained, may be expected.

## NOTES ON CERTAIN LESIONS OF THE NERVOUS TISSUES OF FREQUENT OCCURRENCE IN THE BRAIN OF THE INSANE.

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### *Morbid Affections of the Medullated Nerve-fibre.*

A WELL-KNOWN lesion of frequent occurrence and of grave import in the brain and spinal cord of the insane has been termed Colloid degeneration, and its pathogenesis, as established by Dr. Batty Tuke and others, relegates it to the chapter of diseases of the connective framework, or neuroglia. I do not intend here to dispute this doctrine of its origin, but wish to direct attention to the fact of the frequent occurrence of certain morbid bodies in the brain and cord, which if not absolutely identical with the so-called colloid products, resemble them so closely that it appears impossible to discriminate betwixt the two; and yet the lesion to which I refer, far from being indicative of either a sclerosis or a degeneration of connective elements, is without doubt a product of morbid action in the medullated nerve-fibres. This lesion consists essentially in the presence of oval or spheroidal bodies, perfectly homogeneous in structure, clear, pellucid and colourless, devoid of concentric markings, but frequently exhibiting a tendency to marginal crenulation. These bodies vary in size, their long diameter being usually  $13\mu$ . to  $27\mu$ ., but attaining in some positions a magnitude of  $40\mu$ . They are unaffected by the iodine and sulphuric-acid test and most of the ordinary reagents. They resist carmine staining, but assume a light pinkish hue with hæmatoxyline dye. From the above description it will be seen that they may very readily be mistaken for what have been termed colloid bodies.

That they differ in many respects from the lesion designated miliary sclerosis is proved by the fact that they exhibit no reaction with nitric acid; that they are perfectly translucent, and cannot be seen by the naked eye under reflected light, as is the case with the nodules of miliary sclerosis; and lastly, though the size of the larger bodies and their crenulated margins give them a slight resemblance to the latter, they differ in being perfectly translucent, and never exhibit a fine fibrillated stroma. In every respect they most closely resemble those morbid products termed colloid bodies, and yet, if truly so, they must claim an origin wholly different from that usually assigned to these morbid formations. Their true pathogenesis is established upon the following ascertained facts:—

1. They vary in size in different regions of the cerebro-spinal centres.
2. Their distribution is limited to the region of the larger medullated nerve-fibre.
3. No structural analogy can be traced in any stage of their history between these bodies and the elements of the neuroglia.
4. Direct connection with medullated nerve-fibres is to be detected.

1. The variation in size is important to note, as it bears a direct relation to the varying diameters of the medullated nerve-fibres. In the medullary strands immediately beneath the cerebral cortex their diameter averages  $13\ \mu$ ., the largest very seldom reaching a magnitude of  $27\ \mu$ . In a case which occurred at the West Riding Asylum, in which the onset of the attack was marked by acute delirium, intense cephalalgia, and other symptoms of meningitis, followed by symptoms indicative of a subacute cerebritis, with utter muscular prostration and a rapidly fatal termination, these bodies, supposed to be of recent origin, were found scattered in myriads through the white matter of the convolutions, all of them about  $13\ \mu$ . in diameter, closely resembling colloid bodies, and yet, as will be shown farther on, distinctly from originating the nervous elements. When met with, on the other hand, in regions

possessing nerve-fibres of greater diameter, as in the medulla oblongata, they are found to vary greatly in size, the average dimensions being  $27\ \mu$ ., the larger bodies being fully  $40\ \mu$ . in their longest diameter. This relation of their dimensions to the diameter of the medullated fibre is significant.

2. In all cases where these bodies presented themselves one very striking fact was noted—viz. the strict limitation of these morbid products to regions of the larger medullated fibre. Sections through the gyri invariably showed them to be scattered profusely through the medullated strands, advancing close up to the deepest or spindle layer of the cortex, where they terminate *abruptly* at the commencement or within the confines of this stratum. In the neighbourhood of the ganglionic layer, of the third or large pyramidal and of the second or small pyramidal layer, they were not met with. In the first layer of the cortex they are again often met with, but very definitely limited to its outermost zone, i.e. to the region of the peripheric strands of medullated nerve-fibres which are known to traverse this superficial portion of the first layer just beneath the pia mater. The fact of their limitation to the outer zone of the first layer and the lowest layer of the cortex, whilst the several intervening strata in which the non-medullated fibres predominate were wholly devoid of these formations, is also another feature strongly significant of their affinities.

3. In no case have I been able to detect any intermediate stage whereby they could be referred to morbid changes occurring in the nuclear and cellular elements of the neuroglia. There was usually no proliferation of perivascular or neuroglia elements, nor was there an increase observed in Deiter's corpuscle such as would indicate morbid changes occurring in the connective. Occasionally a nucleus appeared on the surface or border of these bodies, but it could always be regarded as extraneous to the morbid formation and accidentally superimposed. A connective origin has been claimed with much plausibility for the lesion known as miliary sclerosis, and these bodies have been stated to have been traced in all their stages from the swollen nucleus up to the perfectly characteristic form assumed by the patch of miliary sclerosis. In

face of the strong evidence adduced in support of this view I should hesitate to raise a dissentient voice, yet I must affirm that the strange limitation above noted is also recognisable in cases of multilocular miliary sclerosis which I possess. This fact I consider of sufficient significance to warn us fully to reconsider the pathogenesis of this obscure affection, and to study any relationships which they may possibly possess to the morbid products above referred to.

4. So far the evidence deduced, however strongly in favour of the origin of these bodies from medullated nerve-fibres, is of a purely presumptive character, and as such could scarcely be accepted as conclusive were it not for more direct evidence such as was afforded by the case above alluded to. Here these bodies, when carefully examined, were found to exhibit an almost invariable connection with a medullated fibre. Frequently the larger nerve-fibres were mapped out along their course by a series of such bodies forming lateral projections, compressing the axis cylinder or wholly obliterating it. In the smaller nerve-fibres the swelling involved occasionally the whole fibre, appearing like an aneurismal dilatation; the axis cylinder terminating abruptly at either extremity. Similar appearances to these were also presented by the lesions found in a case of glosso-labio-laryngeal paralysis. The fibres of origin of the hypoglossus arising from the nucleus near the floor of the fourth ventricle, as well as the decussating fibres of the raphe, being strewn along their whole course by great numbers of these morbid products, I therefore regard these morbid bodies as resulting from segmentation of the myelinc ensheathing the axis cylinder, and trust I may in a future article be enabled to record the results of further researches into their pathogenesis. In the meantime the object of these remarks will have been fulfilled if they induce others to examine more closely these morbid products, which have been, I believe, too often relegated to the chapter of degenerations of the connective series.

#### *Fuscos or Pigmentary Degeneration.*

This degeneration of the nerve-cells of the brain and spinal cord is one of special interest to the pathologist, as it repre-

sents one of the most frequent of those retrogressive changes to which these nervous elements are subject. It is recognised as being generally associated with those morbid conditions which induce frequent and excessive engorgements of the vascular tracts of the cerebro-spinal system, and hence is seen in most cases of general paralysis, epileptic mania, and senile mania. The constant accompaniment of the fuscous degeneration, therefore, is the coarse and distended blood-vessel, the swollen perivascular sheath bulging irregularly along the course of the blood-vessel and obstructed by accumulations of leucocytes and deposits of hæmatoidin crystals. This lesion has been often described, as it presents itself in sections obtained from hardened chrome preparations, and it may not be amiss to describe here its features as presented in perfectly fresh sections of brain obtained on the freezing microtome. The nerve-cells of each individual layer of the cerebral cortex may be involved in this change, yet the lesion is best studied in those large nerve-cells found in the mid-cortical regions, especially of the motor area of the brain. These elements are in their *healthy state* often somewhat pigmented, although by no means so constantly and extensively as the multipolar cells in the anterior cornua of the cord. With the advent of degenerative changes, however, a notable alteration in the contour of the cell occurs. It becomes swollen, tumid, and eventually even spheroidal in contour, the invaded tract of protoplasm being usually at the base of the cell, but frequently found limited to the borders or the apex. The appearance of the degenerated portion varies in hue from a light fawn to a bright yellow or brownish-yellow, it has a roughly granular aspect, and is unaffected by ordinary staining reagents, contrasting therefore very strongly with the stained and uninvolved cell contents. Its progress, as exhibited by cells in various stages of degeneration, exhibits the following points of interest:—

- a. Retraction of the unaffected protoplasm.
- b. Displacement of the nucleus.
- c. Sclerotic induration of the investing boundary.
- d. Exposure of the *fibrous* structure of the cell.





Fig. 1.

Illustrative of the Progress of Pigmentary Degeneration  
of the Ganglionic Cells in the Cortex of the Brain.

x 280



Fig. 2.

Nerve cells of the Third Layer of the  
Cortex Cerebri—Epileptic Idiotcy.

x 280.



In Fig. 1, *a*, we observe a cell which exhibits this retraction of its protoplasmic contents before the advance of the degenerated portion which is swollen by its imbibition of hæmatin.<sup>1</sup> This retraction gives rise to the appearance of bays (*c*) bounded by fibrous extensions from the unaffected protoplasm to the confines of the cell. At *b*, Fig. 1, a similar condition is observed, involving the summit of the cell. In all cases the nucleus retreats before the invading lesion, the cell presenting all the features suggestive of a morbid and excessive imbibition from without. Should the base of the cell be first involved, the nucleus will eventually be driven up towards the apex process—or towards the base, if the summit be first affected. If the degeneration takes its course from the side of the cell along its transverse axis, the nucleus will be compressed, and even flattened, against the opposite side of the cell. This eccentricity of the nucleus occurs in all cases, its exact position being ruled by the site of the pigmented mass. The cell wall surrounding the degenerated protoplasm becomes in advanced cases indurated and sharply defined, forming a dark irregular border, often much distorted, and presenting the appearance already described by Meynert and Lubimoff as a sclerosed swelling of the cell. With this thickening of the cell wall we often observe the presence of a sharply-defined cincture constricting the cell, and separating the pigmented from the unaffected protoplasm. The appearance, however, to which I would direct special attention is seen at an early stage of the disease, especially in sections obtained from frozen brain. It consists in the exposure of fibre-like continuations of the healthy protoplasm with the processes given off from the diseased area of the cell (Fig. 1, *a*, *d*). These processes pass through the degenerated mass, becoming lost in the retracted and healthier protoplasm. Such a condition I have now frequently observed, and I believe it lends strong confirmation to the views held by Beale and others, that the processes of these cells which apparently originate at the periphery really pass into the substance of the cell, and

<sup>1</sup> In the figures given illustrative of this morbid change of the nerve-cells the degenerated portion is left unshaded, the shaded portion being the healthier protoplasm stained by aniline.

emerge at a distant pole. The protoplasmic matrix in which these fibres are therefore imbedded is less resistant to the encroachment of the morbid change than are the nerve-fibres themselves. The protoplasm becomes pigmented and degenerated, and refuses to stain with the aniline dye, whilst the fibres remaining long unaffected become deeply stained of a bluish-black, and hence distinctly exposed to view. Eventually these fibres also become involved in the degenerative changes, their apparent origin from the cell boundary being marked by a thickened dark-stained nodule. In Fig. 1, *d*, I have represented an appearance not unusual in this disease, the processes where they leave the cell emerging from thickened rings in the cell wall. The persistence of the axis-cylinder process has, I believe, been noted by Mierzejewski, and I can fully endorse his opinion that this process is especially resistant to the degenerative inroads of this lesion. In cases of extreme senile atrophy this process has exhibited a remarkable immunity from degenerative changes, being readily traced, after acquiring its myeline investment, into the strands separating the columns of spindle cells; and yet the apex process would often be wholly absent, or terminate abruptly just above the cell in a linear series of coarse granules, which appeared to map out the former course of the fibre. Again; the diseased cell, whilst exhibiting its basal process intact, would often present itself as a swollen globular body, from the thickened periphery of which arose numerous extremely attenuated processes, terminating abruptly close to the cell, and giving it a peculiar spinous aspect. Vacuolation of the cell is by no means an infrequent accompaniment of these changes. Does the nucleus suffer during the march of these degenerative changes? Its frequent absence establishes the point of its ultimate destruction by the invading lesion, but the actual character of the change which it undergoes is somewhat obscure. We however frequently observe the appearance of oval or circular areas within its structure, which are paler than the surrounding deeply-stained portion, or at times wholly unaffected by the colouring reagent employed. Several of these colourless zones may be apparent in each nucleus, and they evidently indicate a change in constitution of the

nucleus. These degenerated portions do not assume the yellow pigmented aspect.

*The Nerve Cells in Epileptic Idiocy.*

Few opportunities have been afforded me of examining the brain of idiots at the West Riding Asylum, but those cases studied by the fresh method of preparation have all afforded most noteworthy and constant features. These are as follows :—

- a. Spheroidal or pyriform contour of cells.
- b. Marked eccentricity of nucleus.
- c. Coarse granular contents of cells.
- d. Pigmentation extreme, but not a constant accompaniment of the other features.
- e. Great paucity of branches.

It was the second and third layers of the cortex which presented the above characters in a striking degree, the cells of other layers not differing so much from the normal condition except as regards their deficiency in number and extensive pigmentation. The cells of the second and third layers had all lost their usual pyramidal and irregularly angular form, and were universally spheroidal, slightly drawn out towards the apex process, which was invariably present, and deeply stained. Immediately at the junction of this process with the cell the nucleus was situated, and although occasionally the nucleus took a lateral or even basal position, this was very rarely the case, the terminal or apical position being an almost constant feature. The swollen cell could never be stained, except a very restricted zone of protoplasm which occasionally surrounded the nucleus or clung to the sides of the cell, in which latter case the cell had the appearance of clear translucent contents surrounded by a dark thickened border. As to the unstained contents of the cell, they were *usually* of a bright yellow tint, but occasionally colourless and translucent. The contents were coarsely granular, and with high powers were resolved into oval or spheroidal bodies, varying in diameter from 2  $\mu$ . to 4  $\mu$ ., highly translucent, and, as above stated, either colourless or of a bright yellow tint. The whole

cell was closely packed with these granules from the apical to the basal extremity of the cell. It was always observed that when these granules were colourless, intervening fragments of protoplasm occurred, forming stained boundaries betwixt the granules. In a few exceptional instances the cell was more elongated in contour, the nucleus basal in its position, and healthy-stained protoplasm was found beneath the apex process—nucleus and protoplasm being apparently forced asunder by the new material intervening. The deficiency of cell processes was a most striking character in all these cases of idiocy. Most of the cells exhibited the apex process only; others showed one or two secondary branches; but very few possessed so many as four branches, and these were remarkably fine and attenuated. The appearance presented by these cells, magnified 315 diameters, is represented in the accompanying plate.

## ON THE BALANCE OF PRESSURE WITHIN THE SKULL.

BY JAMES CAPPIE, M.D.

IT being admitted that the brain is the organ through which mental acts or states are more immediately produced or revealed, the working condition of the former becomes the correlative of the existence of sensation, memory, thought, and volition. The working condition of the brain depends on a certain standard of structure, and a certain relation to the circulation and to other structures and agents. The more that structure and these various relationships are understood, the better qualified shall we be to trace how far any given mental act or state is related to any special sphere or mode of activity in the nervous centre, and *vice versâ*.

In studying the causation of mental phenomena we need not be deterred by the fact that our knowledge, at the best, is likely to be always remote. It may not be the less positive so far as it may pretend to go. We must ever be content to accept the fact of consciousness as ultimate. The physiologist will never be able to perceive why the brain's activity should be associated with its manifestations. Yet, if a correlation be assumed, he may be able to determine why one form of consciousness rather than another is present; why it is present with a felt amount of intensity; or why in certain circumstances it is obscured or suspended. The nexus between molecular activity of brain and activity of thought and volition may for ever remain "unthinkable," but many of the modifying conditions of the molecular activity may certainly be determined, and the remote but necessary influence of these on the mode and intensity of mental action may thus to some extent be recognised.

In the present essay I wish to give prominence to some of the more general conditions in which the brain is placed—to trace a relation between the organs' mode of working and certain peculiarities in its physical surroundings. I shall have no experiments to detail, nor anything new to reveal in regard to structure. I shall simply apply well-understood principles in physics toward explaining some facts in encephalic physiology, and shall have to depend for the correctness of conclusions on the appositeness of the analogies adduced, and the coherence of my argument. I am aware that in these days of physiological laboratories, when every inference has to be tested by an appeal to the senses, such a plan may not be allowed a high place in the methods of exact science. But in addition to the fact that it is only to a limited extent the general practitioner can avail himself of the more exact instruments of research and experiment to advance physiology, it is possible that some subjects may, at least up to a certain point, be quite successfully treated by broad views being taken of general relationships. In every phenomenon there is of necessity a chain of causation, but the revelations of minute analysis are too frequently only isolated links. These furnish interesting facts rather than explanations. No analysis of sea water would ever enable us to explain the ebb and flow of the tides. It may be, therefore, that by holding some vital phenomena at arm's length, and taking a bird's-eye view of palpable relations, the order of combination and sequence which constitutes causation may be recognised more readily than by atomic or microscopic analysis of the instruments concerned in their production.

It is becoming trite to remark that in the development and activity of everything living, its "environment," "surroundings," or "medium" has an influence only second in importance to that of special structure.

The physiological bearing of the brain's surroundings does not seem to have received the attention it deserves. The skull has been regarded simply as an organ of protection, and less importance is now attached to the peculiarities of the encephalic circulation than was the case many years ago.

Restricting ourselves in the meantime to the skull, much

has been written to show how well adapted its structure is to serve as an organ of protection. By its form, and by the firmness and elasticity of its walls, it combines moderate weight with great power of resistance, and thus, while not a burden to the individual, it secures the delicate structure of the brain against physical injuries.

It is more than probable, however, that the mechanical properties of the skull must have an important effect in influencing the physiological action of the brain itself. If its osseous walls are efficient in affording resistance to agents from without, they will no less exert *repression* on the forces operating within its cavity. The fact that there is a rigid limit to the brain's expansion cannot be without significance.

That in certain circumstances there is a decided tendency to expansion of the brain is well known. When it has been so exposed from injury of the skull that its behaviour can be observed, it has always been noticed that while during sleep the organ tends to sink or retire from the inner surface of the skull, in wakefulness it expands, and not only fills the whole cavity, but it may protrude beyond the aperture. Some forces are at work in the latter state which are quiescent or less powerful in the former. The tendency of these is to make the brain increase in bulk. If its rigid envelopes were suddenly to give way when, for example, severe muscular efforts are being made, the momentum of the blood would cause the vessels to be more completely filled, and the organ would instantly expand beyond the bulk it could possibly occupy while they were entire. To any tendency of this sort, however, the walls of the skull oppose an effectual barrier.

Assuming, then, that when the brain is active its function is exerted under a state of physical repression or stress, we have to consider how such a circumstance may operate in modifying the results of activity.

In physics we have abundant examples of the behaviour of energy under a state of repression. Its tendency is to explosion; or, if liberated in a special direction, to produce intensity of discharge. The utility of the steam-engine, the rifle, and the electric telegraph depends on our being able to control the liberation of certain forces under stress.

From the analogy of physics, then, we may reasonably suppose that whatever energy the brain is able to produce or liberate, it must be more concentrated and sustained, more readily or more completely directed into some special channel, than if it were diffused in all directions in its very production. Or, if we look at the mental correlatives, we may infer that sensation and perception are more exact and acute; thought more coherently sustained; efforts to produce muscular movements stronger and more accurately directed, on account of the physical restraint to which the organic substratum is subjected.

Of course if the brain as a whole requires some counterpoise or fulcrum to insure a required intensity in the discharge of its energy, the same law must apply to individual portions of the brain. Each local or limited area of ganglionic structure which may have specific duties to perform must receive a background of resistance or support from other structures, and increase or failure of this support will have a modifying effect on the result.

It becomes at once obvious, then, that a knowledge of the sources of internal pressure, and of the conditions by which it is modified, is necessary to enable us to understand an important department of encephalic physiology.

The immediate sources of pressure will be similar to those in other parts of the body. There is, in the first place, the expansive pressure of simple growth. When the molecular elements are multiplying or developing into structures more cellular, they attract, with more or less energy, materials from the neighbouring blood-vessels; and in assimilating these an actively expansive force is brought into play, the aggregate amount of which will depend on the rapidity of the process, and the area over which it extends. It is no part of our plan, however, to take cognizance of the intracranial pressure produced from this cause, although, especially in diseased conditions, its consideration is at times of the highest importance.

The second and, so far as the present inquiry is concerned, more important source of pressure is the movement of fluids. We assume that the mechanical force so exerted is in accordance with the ordinary laws of physics. The greater the bulk



and the more rapid the movement, the stronger will be the pressure. Applied to the circulation within the skull, this principle will be equally true in regard to individual vessels or to clusters of vessels.

From this restricted point of view as to the origin of intracranial pressure, its balance will depend simply on local alterations in the distribution of blood through the vessels. Every change in its circulation will be accompanied by a change of pressure in or on the brain.

Our next object, then, must be to determine on what conditions any alteration in the intracranial circulation may depend.

The principal factors concerned in the supply and distribution of blood through the brain are—1, the action of the heart on the general circulation; 2, the action of the vaso-motor nerves on any of the branches of the circle of Willis; 3, the molecular movement in the brain tissue; and 4, the pressure of the atmosphere opposing the flow of blood from the venous sinuses.

In considering on which of these conditions local alterations within the skull occur, the action of the heart may be kept out of view. The ratio of that action will be the same to the whole cavity, and any change of impulse will equally affect all the vessels. Local modifications must be caused by local conditions.

The conditions which will most immediately affect the local distribution of blood are the calibre of the feeding vessels and the activity of the molecular movements. So much sympathy exists between these that they should be considered together. They are so intimately joined that it is impossible to say where causative influence begins or ends. If any distinction, however, is to be made, then greater importance must be attached to the agitation of the molecules. Nothing definite is known as to the action of the vaso-motor nerves on the arteries within the skull. The result of experiments has been negative or contradictory.<sup>1</sup> But we can conceive of the movements of the tissue-molecules acting on the circulation in

<sup>1</sup> See 'Physiology and Pathology of the Sympathetic System of Nerves.' By Drs. Eulenburg and Guttmann. Translated by Dr. Napier, 'Journal of Mental Science,' July 1878.

various ways. Their agitation may communicate its infinitely rapid waves directly to the blood; or the reaction between the blood and the tissue may so modify the attraction of the former for the capillary walls, that the sphere of greatest activity in reaction constitutes the "line of least resistance" along which fluids under pressure must of necessity tend to move; or the molecular agitation may affect the afferent nerve of a vessel—controlling ganglion, which again transmits an influence to the small vessels. It is more likely that any or all of these modes are correct than that any ganglion by itself should have some intuitive perception of the wants of the tissue. Practically, therefore, at all events in the normal state, we may also leave the action of the vaso-motor nerves out of view, and restrict ourselves to the influence which the molecular movement itself possesses of modifying the circulating current.

From the biological point of view, then, the molecular movement in the brain tissue and the movement of blood in the capillaries cannot be separated. Anatomically, such a separation is difficult; physiologically, it is impossible. As an organ which produces and discharges energy, the brain is as much dependent on its supply of blood as the burning fuel is dependent on a current of air: and, again, the flow of blood in the capillaries depends as much on activity in the molecules, as the draught of air through the burning fuel depends on the act of combustion. Check the blood-current, and the agitation of the molecules, with the function involved in their activity, is at once suspended or modified; stimulate the molecular movement, and the blood is transmitted with greater energy. The physiology which would ignore this reciprocal action takes a retrograde step of at least half a century. Even as a "working hypothesis" no amount of precision in describing changes in the histological elements of tissue would ever make up for its loss.

In regard to the influence of the atmospheric pressure in modifying the encephalic circulation, that I consider to be one of very great importance.<sup>1</sup> It results from the physical

<sup>1</sup> See 'On the Relation of the Cranial Contents to the Pressure of the Atmosphere,' *Edin. Med. Journal*, Aug. 1874.

qualities of the cranium, which only permit the contents of the cavity to be affected through the blood-vessels which enter or leave the bony walls. It tends to oppose the exit of blood from the venous sinuses, and the backward pressure so occasioned will, of course, be exerted on the internal surface of the venous vessels through their whole extent. It will thus assist in preventing the block in the entire encephalic circulation that would be caused if the arterial impulse were to press the brain directly against the dura mater. The immediate physical result, as we shall afterwards more fully explain, will depend on the support the vessels receive on their external surface. If the forces causing brain-expansion be relaxed, the veins will be distended; and thus a certain amount of tension is at all times sustained in the brain tissue. On the other hand, if the brain tends to expand, some room is gained by the veins being compressed and their contents lessened.

Assuming, then, that molecular agitation is the dominant factor in modifying the movement of blood—that its intensity and sphere condition the rapidity and direction of the circulation, we are in a position to discuss some details as to the balance of pressure within the skull.

When the brain from a condition of rest, as in sleep, is roused to activity, the immediate effects are to increase the agitation in the molecular structures and to cause more rapid blood movement. This, of course, involves increased pressure within the capillaries, and the result, as we have already seen, is to cause an expansive tendency in the brain itself. To this tendency two important checks are opposed. These are, in the first place, the rigid limits presented by the cranial walls, and to which we need not further allude. The second check is the peculiar structure of the pia mater.

This membrane presents a curious interlacement of arterial and venous vessels, the physiological meaning of which has not received the attention it deserves. Its connections with the brain are entirely capillary. The large mass of the brain is fed by arteries which from the first penetrate its substance in a state of capillary minuteness; the veins also leave the nerve tissue as capillaries, and in the pia mater they coalesce to form large tortuous vessels. Now, if expansion of the brain

were to occur beyond a very limited extent, a process of self-strangulation would take place. The circulation itself would be stopped. The first effect must be to compress the veins, but this compression cannot be to a greater extent than will allow whatever amount of arterial blood which enters the skull to be transmitted onwards. The result will be to increase the tension in the brain itself. The blood pressure will be expended in rapid movement.

To this tension all the fluids and solids within the skull will be alike subjected. It will not be greater at one point than at another. The area being limited and full, and so much of the contents being fluid, the important law of pressure being transmitted equally in all directions will here hold good.

A moment's consideration will enable us to perceive how such a fact is to be reconciled with the statement previously made, that every change in the intracranial circulation involves a change of pressure in or on the brain. We have simply to bear in mind the distinction to be made between active pressure and "stress,"<sup>1</sup> or tension.

In a glass globe where gold fish are in active motion, the currents or agitation in the water can never so affect the globe itself as to make it tend to move in one direction or another. If it were fixed on wheels on a smooth table, "even were the table a lake of ice and the wheels extremely delicate, we should find that the globe would remain at rest."<sup>2</sup> Whether the currents in the water be strong or weak, the pressure on the sides of the vessel will be equalised through its whole circumference. Throughout any particular level the amount of stress will be the same. But within the area of uniform stress, the disposition of movable bodies or particles will depend on the rapidity and direction of the currents, and thus, in the case before us, the movement and position of the fish may vary every moment.

So, within the skull, whether stress be stronger or weaker, it must be equalised throughout the cavity; but the direction

<sup>1</sup> "The word 'stress' as an equivalent for 'action and reaction,' and as a generic name for pressure, tension, &c., will save future generations a great deal of trouble."—Prof. CLERK MAXWELL, 'Nature,' Aug. 20, 1874.

<sup>2</sup> Prof. Balfour Stewart, 'Conservation of Energy,' p. 9.

of the blood currents, and therefore the disposition of the movable solids, will be subject to infinite variation, and there will be no end to the modes in which the action of the brain itself may be modified from this cause. The law of pressure by fluids in motion—as affected by volume and velocity—must here come into play. As the blood is directed in greater quantity or at a more rapid rate through particular vessels, the active pressure will there be greater or less. Then, of course, the brain itself is not a fluid body. Its cell elements and fibres may be kept closer or more apart, according to the disposition of the fluids by which it is surrounded and saturated. As it can readily be understood that a certain compactness, or, it may be, freedom to move, may be necessary for proper functional action, the intensity or direction of the currents may, from their purely mechanical effect, stimulate, modify, or suspend activity through the whole organ, or in limited portions of its mass.

Recurring now to the case of the brain passing from the rest of sleep to the activity of wakefulness, we have to consider in what manner the intracranial pressure is modified. During sleep the brain circulation is quiet, and stress within the skull is weak; as activity sets in the molecular agitation and the increased impetus of the circulation cause the stress to be augmented. But something more than a change in the intensity of intracranial stress must take place. The balance of active pressure is also greatly altered. To show in what manner this occurs we may reverse the order in which the changes succeed one another, and see what happens as sleep comes on.

From exhaustion, or some other cause, the molecular movements become less energetic, and simultaneously the hold of the capillaries on the arterial blood is weakened. But there are other physical agencies at work within the skull, and the action of these cannot remain unaffected by such a change. For example, we have seen there is at all times a special stress within the veins. The combined cardiac and capillary forces urge more blood into them; the passive resistance of the atmospheric pressure tends to keep back that blood within the skull; and, lastly, the expansive action of the brain tends to

obliterate the vessels themselves by pressure on their sides. Of these forces, that caused by the atmospheric pressure varies least in amount. We have therefore to do with a simple problem in the composition of forces. If one or two be relaxed, the third must bear with greater effect. Thus, if the forces which cause brain-expansion act with less energy, the backward pressure in the veins must act with more decided effect. The flow of blood from them will be retarded so long as the equilibrium tends to be disturbed. When the new equilibrium is gained the vessels will be more distended than can possibly occur during a more active state of the brain.

The same change may be expressed in another way. If the brain tends to retire from the inner surface of the skull (and that it does so during sleep is the unanimous testimony of all who have observed its behaviour through an opening in the bone), it will exert a suction effort on the blood in the veins. It is impossible for suction to be exerted on the cerebro-spinal fluid in the spinal canal, for the contents of this cavity are also removed from the direct action of the atmospheric pressure.<sup>1</sup> A new balance of the circulation is therefore established; less blood circulates in the capillaries of the brain, and to an exactly corresponding extent more must be held by the veins. If a survey be taken of the various conditions involved, it will be seen that this change is a *physical necessity*.

If we are now allowed to assume that when the brain passes from rest to activity, or from activity to rest, a change occurs in the balance of its circulation, then, in accordance with the principle we have insisted on, the balance of active pressure within the skull must also be altered. This change of pressure will of course affect the disposition of the brain tissue, and must have an influence on its activity of function. During wakefulness, when the arterial and capillary circulation is most active, the pressure is outwards. It is expansive, and the reaction against the cranial walls causes the stress through the whole cavity to be increased. Nervous energy is liberated,

<sup>1</sup> I have elsewhere ('The Causation of Sleep') fully discussed the improbability of the cerebro-spinal fluid taking the place of the diminished cerebral circulation in healthy sleep.

and the result is some psychical or motor change. During sleep the venous circulation preponderates, and the pressure is from the surface inwards. The amount of blood within the skull may remain as before; indeed, I believe that in healthy sleep it must remain almost exactly the same. But a larger proportion of it is now external to the brain mass, and its movement there must react physically on the brain. The latter is more or less compressed. When the molecular movement began to flag, the capacity for active function was by that very fact also weakened. But this change of pressure still further interferes with functional capacity. When it amounts to a certain degree, function is suspended—the brain for the time fails to subserve the manifestation of the higher mental states.

We thus seem to find that, in the physical changes which accompany rest—in the more sluggish molecular movement with (as a necessary consequence) lessened intracranial stress, altered balance of the circulation and altered balance of pressure—we get a physiological explanation of sleep itself. We are furnished with a mechanism which secures a rest to the brain sufficiently lengthened to allow restorative changes to occur. No fact in brain biology has been better ascertained than that compression of the organ produces unconsciousness, and this first condition of sleep we find in the distended veins of the pia mater. Then, the whole agency being under the control of the molecular activity, the pressure may be graduated infinitesimally, so as to produce the lightest slumber or a state of heavy stupor. Further, if a stimulus be applied to the brain, the whole series of conditions can be changed *at once*. In this important respect the above theory of sleep contrasts with any other which places the cause in a change of nutrition alone, or in the action of some vaso-motor centre. We need not wait till superfluous cerebro-spinal fluid has been got rid of. The increased capillary and diminished venous circulation will be simultaneous.

The next step of our inquiry should now be to consider some questions relating to the balance of pressure through the brain itself. Assuming that the more immediate seat of active function is also the sphere of greatest vascular activity, we

should likely find that the activity of one part of the brain may affect the function of other parts in various other ways than by direct connection through commissural fibres. In one case, function may be suspended by a derivation of blood from its vessels, as when impressions on a sensory nerve are not felt on account of the mind being absorbed in a train of thought; in another case, an increase of energy may be occasioned from the steadier fulcrum-like support afforded, as when the intensity of muscular contraction is increased by the simultaneous action of other muscles; in a third case, the mechanical impulse of pressure may act as a direct stimulus, as when a sudden noise causes one to start, or, indeed, as may possibly occur in the causation of voluntary motion. Into this wide field, however, we cannot enter in the meantime.



## Critical Digests and Notices of Books.

*Manual and Atlas of Ophthalmoscopy.* By W. R. GOWERS, M.D.,  
F.R.C.P. Churchill, 1879.

It would be difficult to light upon a new book more welcome than a Treatise on Ophthalmoscopy by Dr. Gowers. The difficulties of the eye-mirror are really so great that insistence is our only chance of keeping its value before the body of the profession.

Even among the younger men the number who have gained or who keep up any facility with the instrument is a small one; and of this number, again, many fail to detect ophthalmic disorders, on account of the awkwardness of examinations in private houses. The consultant often sees patients at his chambers, with all the conditions of successful investigation complete. The family attendant, on the other hand, sees them at home, and has to struggle with a chandelier, or to court a gleam from an irresponsive bracket—even a candle-end is hard to find in these days of gas, and every one does not know how good a torch is to be made by twisting together half-a-dozen of the wax tapers used by servants for lighting chandeliers.

Dr. Gowers' book has taken shape from beginnings of a thoroughly practical kind. He tells us his first idea was simply to collect drawings of the morbid eye in medical cases; as these drawings became more numerous and valuable he contemplated an Atlas, and finally the scheme grew into the present illustrated volume. With so much first-hand material, both of pen and pencil, we should have found the publication of a dry atlas rather disappointing. As it is, the clinical record obtains from the plates a great additional help and

vividness, and the reader has the advantage of facts *subjecta oculis fidelibus*, and thus permanently fixed upon his memory. Well-chosen and well-executed woodcuts also are inserted in the text, a large number of which are original, none of them trivial or familiar. The drawings of minute pathology are also among the chief attractions of Dr. Gowers' work.

More immediately attractive, however, are the drawings of the fundus of the eye—some in full colour, others in sepia, all of which are original, and all illustrative of cases recorded. There are twelve sheets of these drawings, each containing from two to six figures. The microscopic sections occupy no less than twenty-eight lithographs. All these drawings—more than eighty in number—are not only published for the first time, but are drawn by the accomplished author directly from his own subjects. The autotypes in sepia seem to be a very happy thought, fulfilling, as they do, all the proper purposes for which they are designed. They have the true economy of effort, expressing just what is wanted and no more. In discarding the name of Atlas, therefore, it will be seen that the author has not discarded the substance.

After the Introduction, the first part of the letterpress deals with the retinal vessels and optic nerve, so far as these have a medical significance. Many remarks in this chapter betray the practised and skilful observer; for instance, the acute observation (p. 11) that increased width of vein does not necessarily mean over-distension, but that wide veins may be underfilled and atonic. In this case the veins, according to the author, have an elliptical section, enclosing thus a smaller lumen than a circular outline. Dr. Gowers says that atheroma is never to be detected in the retinal artery, either during life or on autopsy, though it may in rare cases be the seat of aneurism. In discussing the changes of the optic nerve, the author writes some valuable paragraphs on the issues of inflammation in cases long under observation (p. 47 et seq.); he insists with all other observers upon the now well-known though strange fact that good ordinary vision can persist with full neuritis, even descending neuritis, and he supplies a great omission in my own book, namely a description of the changes in the field of vision, and of colour perception. The gradual

advent of colour-blindness was unknown when I wrote, and although the changes in the visual field were beginning to receive attention, yet work in this direction was then so recent and so incomplete and the bulk of my notes so imperfect in these respects that I could not handle the subject. But the importance of these inquiries is paramount and sometimes of crucial value in diagnosis.

Dr. Gowers offers a new terminology of optic neuritis, and I admit that its causation needed re-discussion. The sections (p. 63 et seq.) on this subject are very able, and treat of the bases of our views of optic neuritis and its relations; we do not find that the subject is worked out to a settlement, but we certainly find it advanced a stage, especially by means of post-mortem investigations.

The second part deals with the ophthalmoscopical changes in spinal diseases. As the reader passes quickly from section to section, he will find with regret that Dr. Gowers unfortunately has to agree with all observers in failing to find any direct reflection in the retina of degrees of anæmia and hyperæmia of the brain. In softening of the brain he finds optic changes to be more frequent than hitherto has been supposed. In tumours, he testifies of course to the frequency of optic neuritis, but fails still to find out the connection between the two events. In some cases the optic neuritis seems to move *pari passu* with the advance of the tumour, in others to take an episodic cause of its own. The size and nature of the tumour seem to have nothing to do with the causation of optic neuritis. Under meningitis, we find still some ophthalmic evidence that this disease, as I urged long ago, occurs in slighter degrees more often than we are aware, and that even when well marked it does not always destroy life. Dr. Garlick has produced some remarkable testimony to the same effect. Under spinal diseases Dr. Gowers says that ophthalmic changes are less common in locomotor ataxy than is supposed, and herein I think he is right. He thinks indeed the percentage will come out even less than fifteen per cent. A curious case is given by the author and Dr. Buzzard, in which optic atrophy and loss of "knee reflex," appeared long before the gait became affected. It seems clear at any rate that the

changes in the eye do not depend directly upon the degeneration in the posterior columns. The extreme rarity of optic atrophy in lateral sclerosis is still attested. I have never found this coexistence; a single case is published by Dr. Gowers; but in it, if the diagnosis be clear, the conditions were far from simple. In disseminate sclerosis I have found optic atrophy more frequently I think than Dr. Gowers indicates, but the disease is not very common in Yorkshire. Of optic changes in injuries of the spine the author speaks little, and well says that in litigated cases the morbid changes are more often in the mind of the observer than in the eye of the observed. Of this I may give an instance. An eminent ophthalmic surgeon, in a "railway case" tried last year at Westminster, found changes in the optic nerves and retinae of the plaintiff so grave in kind that he positively prophesied complete blindness in eighteen months from that date, namely about June 1879. The evidence of other ailment was weak, and Lord Justice Coleridge said frankly that the verdict would depend on the value of this ophthalmic testimony almost alone. Although the two other observers called into the box had been unable to detect any morbid changes in the eyes, the jury naturally feared to defy the confident assertions on behalf of the plaintiff, and gave very heavy damages. The judge proposed that execution should be delayed in order to test the prophecy of blindness, but the plaintiff failed to see the force of the suggestion, and he is, I understand, at present enjoying his fortune with all the advantages which good vision can bestow. Surely such a conflict as this upon facts which ought to be positive enough is anything but creditable to ophthalmic science, and Dr. Gowers, as we have seen, washes his hands of such controversies.

To return to the volume before us. On p. 157 we should have been glad to see some reference to that remarkable malady, herpes frontalis. Of the changes in albuminuric retinitis, Dr. Gowers gives clear account, but adds little or nothing to our present imperfect understanding of this curious event.

Under the head of Diseases of the Blood, we find an interesting and indeed remarkable chapter on the ophthalmic

changes which may follow large hæmorrhages. There seems to be a wide consensus of evidence that the loss of vision in these cases is not functional only, but often depends on actual and visible inflammatory changes in the disks and retina—the loss of sight being often permanent, and sometimes complete. Hæmorrhage from the stomach seems to be the most efficient cause of such permanent impairment of vision. In most cases the ultimate change is one more or less of atrophy. I am bound to say that such terrible consequences, which can scarcely be overlooked, have not been evident in my own practice, but it is needless to add how serious a matter it is. Happily this terrible consequence rarely follows traumatic hæmorrhages or therapeutical blood-lettings. The tendency in pernicious anæmia to retinal hæmorrhages is a different kind of process, and of this malady I have had exceptionally large experience; in my opinion the retinal hæmorrhages occur in the minority of cases, and are when they occur a sign of rapidly advancing or advanced disease. The section on the retinal changes in leucocythemia adds little but the author's valuable testimony to our previous knowledge of the process.

Most physicians who work with the ophthalmoscope have noted optic neuritis standing alone—apparently “idiopathic”—in young women. Dr. Gowers thinks it may follow sudden suppression of the menses in the manner that acute myelitis may do. I may add in like manner that I have lately thought that amenorrhœa may in some cases be the cause rather than the usual mere concomitant of phthisis. In such cases the phthisis has dated from a sudden suppression of the menses in healthy women coming of non-phthical families.

As I ventured to say years ago, Dr. Gowers and Dr. Garlick state that choroidal tubercle is very rarely visible to the eye-mirror; Dr. Garlick searched for it in the Children's Hospital for two years, and found it but once.

The chapter on Lead-poisoning is short, but clear and good; and a good case of optic neuritis due to this cause is given with a drawing of the fundus. Concerning tobacco amaurosis, Dr. Gowers has little to add to Mr. Hutchinson's opinions. It seems probable that amblyopia is caused by the use of certain kinds of tobacco.

A series of test types and a scheme of tests for colour blindness are found in an Appendix, together with some useful hints on ophthalmoscopes and the method of their use. Skeins of green and purple wool are methodically used in Germany, and are better than coloured cards as tests for Daltonism. The series of cases upon which the work is founded and which have supplied materials for the Atlas are put together at the end, and form an invaluable body of evidence and means of instruction. Dr. Gowers has thus well met a real want, he has brought the study of medical ophthalmoscopy up to the present date; he has used old materials judiciously, and original materials extensively and carefully; and he has given us his results in a handsome and well-illustrated volume, of which not only the author and publisher but even the profession in England may be proud.

Medical ophthalmoscopy was established by the Germans, but the work before us completes the claim of England to an equal share in its later development.

T. CLIFFORD ALLBUTT.

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*The Study of Psychology.* By G. H. LEWES. London, Trübner and Co., 1879.

IN accordance with the announcement made at the time of the death of the late Mr. G. H. Lewes, that his loss would not prevent the publication of more of his writings, the first posthumous work has now appeared, under the above title, and forms the first problem of the third series of his well-known critical and constructive 'Problems of Life and Mind.' Though of much less bulk than its companion volumes, it contains much that is instructive, and many readers will esteem it highly among all of the author's works, for it is occupied with questions that are of the highest interest, and are most warmly disputed at the present day; and a critical examination of antagonistic opinions, together with an expression of the views of so high an authority as the author, must needs assign to the work a high and lasting value; and it is worthy of remark that the author's special opinions neither have undergone

material alteration, though more definite and further developed, nor are less enthusiastically supported, than when he first wrote upon these subjects.

He begins by stating that certain preliminaries, common to all sciences, are required by the study of Psychology, viz., definition of the object and scope, the motive, and the method of the search. But, unfortunately, unlike the other sciences, there is still the utmost discord prevalent with regard to the aim of, and consequently the method to be employed in, this one. The chief hostile views are thus described (p. 1): "On the one side stands the ancient metempirical conception of a so-called Rational Psychology, with its deductive method of ontological research. Its adherents, even when condescending to what they call Empirical Psychology, so little regard the data of experience, that they quietly ignore the complex conditions of the living organism, and treat mental facts simply as the manifestations of a psychical principle, at once unknowable and intimately known, a mysterious agent revealed to consciousness. On the other hand, there is an empirical school which professes to confine itself to the data of experience, and to pursue the inductive method, discountenancing ontology and coquetting with physiology. This school keeps up the traditions of a psychical principle, independent of the organism, and of introspection as the exclusive method of research. Of late years there have arisen writers who have tried to effect a compromise, invoking physiological data for one class of facts, and only invoking the psychical principle when physiological data fall short." It is the last party with which the author is most closely allied, and which has made most progress of late; for it can scarcely be doubted that the co-existence of extreme opinions showed the science to be in a transitional state, and that a proper medium would be most efficacious in placing it on a definite and stable basis; but although the author's views are identified with the main current of modern speculation, he protests against certain courses into which it appears liable to drift. The chief cause of the movement has been the steady appreciation of the existence of universal and unfailing laws pervading mental phenomena: introspection, however, showing that

numerous breaks in the chain of causation, on the side of consciousness, prevented their due recognition in that way, the continuity must therefore exist only between processes beyond the range of reflection. Now the aid that modern science has rendered to Psychology has been to show that, instead of being obliged to rely on an *immediate* view of a continuous series of conscious states, which would be impossible, as we can neither extend our range nor shift our point of introspection, we may attain the same end *mediately*, by analogical reasoning. But, for this purpose, it is necessary to adopt a new method of research—to translate the facts of consciousness into terms of another class. The value of this method is the chief point of dispute, and though as yet confessedly in a very immature condition, its use is warmly advocated, though with cautions against its abuse, by the author. The new method follows from the assumption, for long tacitly, though fitfully, made by all, that organic and mental processes are strictly correlated; so that a complete analysis of the one would give an equally complete acquaintance with the other: and then, while some terms of the one series may be beyond our reach, the corresponding members of the other series may be accessible. On these points Mr. Lewes says: "Modern science has revolutionised the question by showing physiology and Psychology to be two aspects, instead of two distinct sciences." Again (p. 76): "Mental state and organic state are only two aspects of one and the same thing, distinct from each other in so far as they are apprehended in different ways, and expressed in different terms." And again: "Objectively we know a sensation as a vital fact or movement, a component in some conscious resultant, which may be present or not; this sensation lies out of introspection, but may be brought within its range experimentally. Thus, much becomes explicable on wider observation than introspection."

The problem being, as before mentioned, threefold, the contents of the book fall naturally into as many parts—occupied with the endeavour to answer the questions respectively—What should we study in Psychology? Why do we study it? and, How are we to do so? Though the main questions can



be thus isolated and clearly stated, we are as yet unable to give a complete answer to all of them; and even such answers as are forthcoming are scarcely susceptible of statement in terms less abstract than the questions. With reference to the incompleteness of even these preliminaries, Mr. Lewes says (p. 4): "The constitution of the science has still to be effected;" meaning thereby the negative process of *circumscription* of a class of phenomena, and its positive fixation by *specification* of the object and method of search: yet "a first approximation" may be made now, by adopting the methods of inquiry already within our reach. In answer to the first question, the author gives as a definition, which he undertakes to justify hereafter: "Psychology is the analysis and classification of the *sentient functions and faculties* revealed to observation and induction, and completed by the reduction of them to their *conditions of existence*, biological and sociological;" "for," he observes, "it is necessary to observe the medium with the organism." Elsewhere Psychology is defined more briefly as "the science of the facts of sentience."

Though careful to state that an analysis of the terms of the definition is not demanded of the science defined, belonging of course to a higher sphere, he goes a little out of his way to explain the nature of mind, in order to elucidate the subject, and make his general argument more easily grasped; thus, he says: "Mind is a form or function of life;" "the relation of mind to life being so plain that no one doubts it, yet so obscure that no one can precisely describe it:" and thus "Psychology is a branch of biology."

In expressing such a view of the filiation of the two sciences, it is remarkable how closely it resembles Aristotle's theory of the soul, according to which mind and life are homogeneous, differing only in speciality; and the resemblance is carried still farther by the partially isolated position he assigns to the most highly specialised human faculties, in correspondence with the "noetic soul" of Aristotle. He even touches upon the metaphysical question of the nature of mind—whether or not it may be regarded as a substance—in order to refute the theory of a psychical principle, for which he says there is no more evidence than for a vital principle or motor principle.

His metaphysical argument may here, for completeness, be briefly summarised, though probably it is already familiar; and it appears to us to contain a confusion between "per-cipere" and "percipi;" the ultimate of knowledge is feeling, which has two aspects—subjective and objective—mind being an abstraction generalised from the sum of the former aspects, the object-world an abstraction generalised from the sum of the latter. Hence he arrives at the somewhat strange conclusion that the subject-matter of Psychology (with sociology) and of cosmology is identical, and has in each case the same two aspects, but while the one aspect is prominent in the former, the other aspect is most prominent in the latter: and biology occupies an intermediate position, neither aspect being throughout the more conspicuous. The theory appears to us to contain a confusion concerning the question of the opposition of subject and object—the author seeming to forget that the contrast persists as vividly in *psychology*, although its *ontological* nature may be disregarded. This seems to be the objection raised by Professor Green to Mr. Lewes' metaphysics in his critique ('Contemp. Rev.' 1878). In contrast to Aristotle's *metaphysical* theory of the soul, Mr. Lewes regards mind rather in its bestowing sentience, than, like Aristotle, a peculiar power, as of a mainspring, upon the organism; but yet the latter opinion seems at least predominant when mind is described as a form of life.

Having declared the generic identity of mind and life, he proceeds to point out the specific distinction between them by discussing the relation of physiology and Psychology; for though both refer to the functions of the organism, yet "Psychology is beyond the province of physiology;" "physiology deals with physical processes, the organic conditions of production; Psychology with the products—processes which are, have been, or may again be conscious."

Here we may notice a few terms much employed by Mr. Lewes, and invested by him with a peculiar meaning, though not all adhered to with strict consistence throughout, in order to express his special views. First is a distinction he proposes and adopts between "Function" and "Faculty," the former connoting a native, the latter an acquired activity.

Next is a similar distinction drawn between "Mechanism" and "Experience;" the former signifying fixed, the latter acquired and variable structure or constitution. A very similar distinction separates Physiology and Psychology; the former being concerned chiefly with functions as the activities of a mechanism; the latter with faculties as the activities of experience. With these definitions we do not feel altogether disposed to agree, the distinction being not sufficiently radical—not of kind, but merely of degree. Further, it seems a pity to divest the word "Faculty" of a useful meaning it already expresses. And also we think "Experience" would be very advantageously used in a wider sense than here proposed, and as it really seems to be used here and there in the work, applied to the total sentient aspect of the organic constitution. "Sentience" is applied, as in the preceding volume, to feeling in its widest sense, "Consciousness" being restricted to introspection or reflection. Just as, in 'The Physical Basis of Mind,' the author repeatedly insists on the whole organism being involved in its every action, as the conditions of its occurrence, so does he frequently maintain here that the whole sentient organism is requisite to every psychical state.

The object and scope having now been set forth, the motives of the study are considered. They are the same as prompt the formation of other sciences—speculative (theological and scientific) and practical. On account of the latter, mind and society have received especial attention because of their great influence on human interests, and their relatively easy modifiability. Among the former, no explicit mention is made of the special bearing of this science on metaphysics, as being the key wherewith to unlock those ultimate problems which confront us, though it is implicitly stated throughout Mr. Lewes' works. By far the largest portion of the book is taken up with the question, How the study is to be conducted? First, the position of the science in the cyclopædia is described, to prove that the same method must be employed in all sciences alike. This assimilation with the other sciences is of comparatively modern origin, mind having formerly been considered as a distinct principle apart from the body.

The same laws are equally applicable to both groups of sciences, because, as previously shown, they are really the same phenomena regarded under different aspects. The author confesses that he has been so often perplexed and vacillating (and we venture to think that he never attained complete maturity) in trying to assign to Psychology a definite place, that he criticises the opinions of Comte, Mill, and Spencer, as the best means of putting forth his own by contrast. Comte, he says, did great good by making Psychology a branch of biology, but committed a great error in despising introspection. Mill, on the contrary, overrated introspection. Spencer, with whom Mr. Lewes of course closely agrees, displeases him in only admitting in a qualified way that the science of mind is only a branch of that of life, and in regarding consciousness as absolutely unique; he also differs on some subordinate points. The next stage of the inquiry is "to specify the method, and register the fundamental inductions," beginning with a chapter on the "Social Factor," whose recognition is the final step in establishing the constitution of the science. This important factor is (p. 76) "not simply an addition, like that of a new sense, which is the source of new modes of feeling; it is a factor which permeates the whole composition of mind." "The organism creates and is in time modified by the social medium." Lewes claims originality in showing the vast range and mode of operation of the social factor, e.g., sentiment and science are inexplicable by the mechanism which physiology treats of; for they involve experience which is possible only to the collective life. Thus society is a factor of the individual experience in a double sense—in providing the matter for experience, as well as the instrument by which this is conveyed, viz., language, on which depend tradition, science, and art.

In acquiring data, both observation and introspection are indispensable; the former to give facts about ourselves, other individuals, society, and history; but these always require to be supplemented by introspection, whose data are *certain*, while those of observation are *exact*; and by interpreting the results of one as terms of the other, certainty and exactness may be combined. But introspection, with numerous other limita-

tions, fails to show us the *conditions* of psychological facts, although it does not deny them; this is illustrated by a chapter on the freedom of the will. Consciousness tells us that we make a selection of one out of many motives; and "this power of choice endows a motive with superior energy;" and "besides the particular motives, we are conscious of a personality, which determines them to be what they are." But from the biological point of view the conditions are revealed, and determinism is plain; self is the incorporation of our past experiences, and bears the same relation to the individual volitions as the organism does to its constituent organs.

In discussing the use of objective analysis, the author warns against falling into the traps of "hypothetical physiology," an error too often committed at the present time by drawing unwarranted physiological conclusions from anatomical facts; and employed ignorantly "to conceal gaps in our knowledge." Objective analysis seeks to complete and verify the data of subjective analysis. It consists in reducing the facts of zoology (including anthropology) and history to their conditions in physiology and sociology; till this is effected, "observation discloses only symptoms, not causes." Next comes a discussion of some importance on the value of observations made on animals, to learn the possibility of a comparative psychology; but the general conclusion is, that though undoubtedly it would be of immense advantage if we could obtain the necessary data, yet these are really quite beyond our reach, inasmuch as our interpretation of the observations of the actions of animals into their supposed sentient states is too much biassed by anthropomorphism; for their feelings, even though homologous with ours, may be widely different when referred to an absolute standard. Therefore we shall never rightly understand animal psychology until we have a tolerably complete human psychology into which to translate it.

As a natural sequence to this argument, there follows an interesting comparison of animal and human attributes, but the author seems rather to make gradational differences obscure homological resemblances. It is certain that the chief qualities of man are wanting in animals; even the senses are not the

same, but similar. Even if the mechanisms were identical, there is much difference in experience; one important result of our more complex experience is reflection or consciousness. Animals have scarcely any sympathetic altruistic impulses except the sexual and parental; and are insusceptible of virtue or vice, wisdom or folly, the pleasures and pains of ideal life; which are all due, through language, to the social factor. By following out the operation of this factor through language, we can at any rate ascertain some of what phenomena are *not* to be found in animals. Their intelligence and passions become transformed into the intellect and sentiment of man. The moral sense is also a social product; though controlled by fear, animals cannot distinguish right and wrong; love of approbation is a great advance on the former sanction, though far inferior to the latter; but in even some human minds, "what is approved of" is the only aim of conduct. Still "the sanction which once was the outside whip has become the inward sympathetic pang." The author seems, however, to imply that egoism is a powerful factor even in the most lofty actions, when he says, "Much right action springs from healthy impulse." Opinion of what is right and wrong has been much influenced by the misguidance of superstition. Conscious judgment and immediate impulse are blended in all proportion, according to the relative influence of heredity and training. "The abstractions right and wrong become, in course of time, centres around which other emotions group themselves."

History affords an immense store of facts, whence we may infer the present conditions of society and the individual mind; it may be looked upon as an experiment instituted by society, just as disease is a natural experiment in physiology. It must always be resorted to, to teach how experience has grown; also, it thereby teaches the laws of the sentient faculties, especially moral and intellectual. The true logic of science is only shown in the history of science. "The principles of art also are only thus to be acquired. The "general mind," much more even than the individual mind, is a historical growth. History also shows the growth of mind, a knowledge of which is as essential as is embryogeny to phy-

siology. Introspection does not suffice, but still the author holds that there is no warrant for Prof. Green's assertion that "the observation by the mind of the mind's genesis is the crowning absurdity of speculation." To us it appears legitimate to go farther than Mr. Lewes, and hold that introspection, aided by memory, can show us the greater part of the genesis of the individual mind. The "general mind" receives a special chapter: it is said to be a factor always implied in psychological discussions, though seldom clearly conceived, viz., the experience of the race in its influence on that of the individual. Every thought belongs to an impersonal reason—Humanity. A solitary man would think, &c., but would not form conceptions. Still the general mind is but an abstraction; not a "*res completa*." The units of society have a common impulse and a common end, common desires and opinions. The individual avails himself of the experience of others because his is so feeble. Civilisation is the accumulation of experiences; and the collective experience is the chief human attribute, it acts on the individual mind through the social institutions of scientific theories, works of art, and especially language. This is virtually the same as Hegel's description of the development of mind. But there is much prejudice and incoherence mixed with sound knowledge; and obscure beliefs are largely mixed with logically assignable premisses in drawing conclusions. Hence we see how great must be the chance of the superabundance of tares occasionally choking the wheat, and can understand the need of Bacon's warning against the four idols, and consider Descartes justified in starting with a universal doubt. A clear conclusion is to be had only in the exact sciences; quite the reverse in the moral sciences, and ordinary reasoning. Still the author remarks that "Hartmann's display of the operation of 'the Unconscious'" is an exaggeration. Conclusions once obtained become "necessities of thought" for the individual, e.g., the rules of arithmetic are now, and the law of evolution will be so in course of time. Still disciples of Kant are not to interpret these conditions of experience as the mental forms which form the essence of his system; but Mr. Lewes' criticism of Kant here appears scarcely as thorough as we could wish. The final chapter is to insist upon the necessity

of analysis and synthesis as implying each other. Just as in former pages the author blames crude materialism, so here he exposes the shallowness of sensationalism; for thought and the totality of experience is the presupposition of every particular sensation; and in dealing this blow to sensationalism, we think the author is doing good service.

We have now given, by free condensed quotations, a rough outline of this suggestive volume; still we cannot pretend to have presented its most delicate features, or even to have accurately shown the author's peculiar views; however, we feel sure that all who are interested in the subject will supply these from the original, and we should be much surprised if they do not look forward with pleasure to the appearance of the next promised volume.

F. L. BENHAM, M.B.

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*Etudes Cliniques sur les Lésions Corticales.* Par le Dr. Henry C. de Boyer. Paris, 1879. 8vo. pp. 230.

IN this very exhaustive and copiously illustrated monograph M. Boyer gives a *résumé* of the methods and results of recent researches on the anatomical and physiological topography of the brain, and reviews the whole of the clinical evidence bearing on the localisation of cerebral functions. He quotes and gives references to 360 cases of lesions of the cerebral cortex. The following are among the chief conclusions which he founds on his analysis of these facts.

That the occurrence of motor affections with cortical lesions depends on the seat of the lesion. Lesions of the following regions are unaccompanied by motor symptoms: 1. On the *inferior* aspect, the occipito-temporal and orbital regions; 2. On the *superior* aspect, the anterior two-thirds of the territory of the anterior cerebral artery, and the whole extent of the territory supplied by the posterior cerebral artery; 3. On the *internal* aspect, the territory of the anterior internal frontal, and that of the middle internal frontal in its anterior half, the cuneus, the posterior half of the præcuneus, and the whole of the temporal convolutions; 4. On the *external* aspect,



the latent region is co-extensive with the domain of the anterior and posterior cerebral arteries. Lesions of the parieto-occipital region may also occur without hemiplegia. Occasionally in connection with lesions of the *pli courbe* oculo-motor-paralysis without paralysis of the limbs occurs.

The lesions accompanied with motor paralysis occupy the region of the fissure of Rolando and origin of the fissure of Sylvius. Aphasia is found associated with lesions (*a*) in the third left frontal; (*b*) the island of Reil of the left side; and (*c*) the left third pediculo-frontal fasciculi. The minimum extent of the lesion causing aphasia is the posterior third of the third left frontal round the ascending branch of the fissure of Sylvius, but especially in the operculum and base of the convolution.

Some conclusions as regards localisation may be founded on cases of partial epilepsy—which never occur with lesions on the inferior aspect of the hemisphere—but with less certainty than from cases of monoplegia or partial paralysis. Of monoplegia we have cases of aphasia, facial monoplegia, brachial monoplegia; more rarely, crural monoplegia. We may also have ptosis, rotation of the head to one side, with or without conjugate deviation of the eyes. Of associated monoplegia we have cases of face and speech, face and arm, arm and leg; but never, with circumscribed lesion, of face and leg, or speech and leg. The arm centre therefore separates that of the face and leg.

These centres are arranged from below upwards from the insula to the paracentral lobule along the fissure of Rolando, and correspond to the descending order of the muscular groups which they set in action. The speech centre is the posterior third of the left frontal; the face centre the base of the ascending frontal and parietal; the arm centre, the middle third of the ascending frontal and parietal; the leg centre, the upper third of the ascending parietal. The cerebral origin of the third pair of cranial nerves perhaps occupies different points of the *pli courbe* and inferior parietal lobule. The centre of rotation of the head is probably situated at the base of the second frontal convolution. These two latter centres, however, are not yet satisfactorily localised.

It is difficult to determine with exactitude the extent of the arm centre, as a part of its territory is common with that of the leg; yet as isolated movements of the arm and leg are the rule, and as brachial monoplegia does occur, we may suppose, either (1) the existence of a centre common to arm and leg, and distinct centres of certain movements of the arm and leg; or (2) the superposition in different layers of the cortical substance of two centres, the one for the leg, extending towards the parietal lobule, the other, for the arm, descending towards the middle third of the ascending frontal; or, (3) a union in some cortical regions of cells for the arm and leg, or of fasciculi, with a maximum for the arm below and the leg above.

There exist two zones which may be termed *neutral*, without prejudging their functions; the one occupying the superior parietal lobule, the other the neighbourhood of the paracentral lobule, that is, the anterior half of the præcuneus and a part of the gyrus fornicatus.

The observations relative to circumscribed lesions of this zone do not serve to determine whether it is motor or not. It is probable, however, that it belongs to the latent zone.

One of the constant anatomico-pathological characters of cortical lesions is the occurrence of secondary degeneration in the lateral columns of the spinal cord. The atrophy of a centre in consequence of arrest of the functions of the limb which it governs cannot be regarded as sufficiently well established. The sensory centres of the cortex appear to be situated in the zone which is latent as regards motility, but we have not yet sufficient evidence to enable us to fix their precise situation. The vaso-motor centres are less certainly determined than the last.

D. FERRIER.

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*Lectures and Essays.* By the late WILLIAM KINGDON CLIFFORD, F.R.S., late Professor of Applied Mathematics and Mechanics in University College, London, and sometime Fellow of Trinity College, Cambridge. Edited by LESLIE STEPHEN and FREDERICK POLLOCK. With an Introduction by F. POLLOCK. 2 vols. Macmillan & Co.

“THOSE whom the Gods love, die young.” It might have been said of those whom the Gods envy, if it were meant as a lament for the cutting short of precious and promising life, and the waste of gifts and faculties which have been called divine. It may be that these gifts appear more precious in the young than in the aged, and that after the wear and tear of a long life the possession of them might be questioned in the very men who in earlier years have appeared the most gifted, and the most blessed. But perhaps the assertion is only one film of the disguises in which sorrow is accustomed to hide itself deep in proportion to the loss it has sustained, and which, if life be worth having, as we believe, has a right to be the greater, in proportion to the amount of precious life which has been cut off.

The subject of this Biography and the author of these Essays might well be said to be one whom the Gods loved, for he was abundantly blessed with their most precious gifts. To marvellous power, facility, and range of intellect, in him was added the exquisite charm of a sweet and genial temper, a sympathising disposition, loving and compelling love, “a merry heart which goes all the way,” and a sincerity which made itself felt and could not be doubted. Probably it was the addition of this truthfulness and trustworthiness to the intellectual light and emotional sweetness of Clifford’s character which so strongly drew comparative strangers towards him, as his biographer mentions. And when to these qualities are added a modesty and simplicity which made him childlike, and a gaiety of humour which made him brilliant, a character is completed which is here lovingly and faithfully portrayed in the affecting biographical sketch of one of his intimate friends.

Intellectually Clifford did not strike one as a giant like his old master Whewell, and the account of his acquirements given by his friend is in these days of universality not overwhelming. He was rather a *preux chevalier* of intellect, reminding one of the qualities of Sir Launcelot when he went disguised to win the diamond necklace at the last tournament ; —it was the grace and versatility of the man which distinguished him from the other combatants. “Nihil tetigit quod non ornavit,” and he did touch and adorn many things. Power alone is not genius ; but power, combined with versatility and a quality for which we can find no better term than notional instability, comes very near to that rare and precious product. There is nothing farther from what we mean by this term than fickleness, or uncertainty ; we mean, rather, a ready faculty of changing the direction and combination of ideas. In some minds, and powerful ones too, certain groups of ideas always hang together ; they never split up and recombine. They are pretty certainly prompted by ideas and followed by other ideas which, if you knew your man you might predicate. But in a man of genius, ideas readily split up and recombine with others, according to the rules of inorganic rather than of organic chemical change, thus producing a greater and more beautiful variety of thought, illustration, and eloquence. Genius has power of quick change in the direction of thought, like that which Clifford showed physically in his gymnastic corkscrew, consisting in “running at a fixed upright pole which you seize with both hands and spin round and round, descending in a corkscrew fashion,” and this ideational instability belonged to him in a pre-eminent degree, though the mental change was mostly from the tortuous to the direct ; it was manifest in his discourse, and is one of the most marked characteristics of his writings.

We think the biographer has not done full justice to the beauty of his literary style in saying : “I do not think he cared much for the use of language as a fine art, though he had a great appreciation of arrangement and composition. His own style, always admirably clear and often eloquent, was never elaborate.” But whether Clifford cared or not, his use of words *was* a fine art, and it would be difficult in English litera-

ture to match his style for the beauty which results from a combination of simplicity, lucidity, and power, more fairly than by likening it to that of Jonathan Swift.

This sketch, however, of a simple life is as interesting and pathetic as keen appreciation and fine characterisation can make it, reminding one of those biographical elegies to his friends which have recently been published by Oliver Wendel Holmes.

The biography is followed by extracts from a few letters, only enough to whet one's appetite for more. The Lectures and Essays which follow represent Clifford rather as a teacher than a debater, or even as a thinker, though the most popular among them are full of originality either of his own thought or of his method of putting the thought of others. Many of them will be full of interest to the readers of 'BRAIN,' as must be obvious from their titles: 'Conditions of Mental Development;' 'Theories of the Physical Forces;' 'Body and Mind;' 'Right and Wrong;' 'The Ethics of Belief;' 'Virchow on the Teaching of Science.' Perhaps the last of these Essays, which we are told was written at one night's sitting, is as remarkable as any of them, as an example of the author's power of placing difficult problems before his readers so that no one can fail to understand, not only the general argument but the shades of probability and degrees of belief, which could rightly be attached to many different propositions. It is of course founded on the well-known argument between Haeckel and Virchow, not only as to what is really proved on the evolution theory, but also how much of it it would be right to teach children; and those who have mistakingly thought Clifford extreme in all his opinions will be surprised to find that he endorses the moderate conclusions of Virchow, and the exposition of the degree of credibility of the two propositions which he agrees to be unsettled, are a good specimen of his power, fairness, and lucidity in the statement of argument. These propositions are: 1st. The Descent of Man from some non-human Ancestor. 2nd. The Doctrine of Spontaneous Generation. But for the purpose of an example of his matter and method, we prefer to give a quotation from his earlier paper on 'Body and Mind.' After having described the physical changes involved in the reception of sensation and its conver-

sion into motion, he thus argues as to whether or not there be any change over, above, and beyond these physical changes.

"Now the question is, Is there any creation of energy anywhere? Is there any part of the physical progress which cannot be included within ordinary physical laws? It has been supposed, I say, by some people, as it seems to me merely by a confusion of ideas, that there is, at some part or other of this process, a creation of energy; but there is no reason whatever why we should suppose this. The difficulty in proving a negative in these cases is similar to that in proving a negative about anything which exists on the other side of the moon. It is quite true that I am not absolutely certain that the law of the conservation of energy is exactly true; but there is no more reason why I should suppose a particular exception to occur in the brain than anywhere else. I might just as well assert that whenever anything passes over the line, when it goes from the north side of the equator to the south, there is a certain creation of energy, as that there is a creation of energy in the brain. . . ."

"It may be conceived that at the same time with every exercise of volition there is a disturbance of the physical laws, but this disturbance being perceptible to me, would be a physical fact accompanying the volition, and could not be the volition itself, which is not perceptible to me. Whether there is such a disturbance of the physical laws or no is a question of fact to which we have the best of reasons for giving a negative answer, but the assertion that another man's volition, a feeling in his consciousness which I cannot perceive, is part of the train of physical facts which I may perceive—this is neither true nor untrue, but nonsense. . . ."

"The mind then is to be regarded as a stream of feelings which run parallel to, and simultaneous with, a certain part of the action of the body, that is to say, that particular part of the action of the brain in which the cerebrum and the sensory tract are excited. . . ."

"But we have to consider not only ourselves but also those animals which are next below us in the scale of organisation, and we cannot help ascribing to them a consciousness which is analogous to our own. We find when we attempt to enter into that and to judge by their actions what sort of consciousness they possess, that it differs from our own in precisely the same way that their brains differ from our brains. . . ."

"The only thing that we can come to, if we accept the doctrine of evolution at all, is that even in the very lowest organisms, even

in the *Amœba* which swims about in our blood, there is something or other, inconceivably simple to us, which is of the same nature with our own consciousness, although not of the same complexity; that is to say, we are obliged to assume, in order to save continuity in our belief, that along with every motion of matter, whether organic or inorganic, there is some fact which corresponds to the mental fact in ourselves. The mental fact in ourselves is an exceedingly complex thing; so also our brain is an exceedingly complex thing. We may assume that the quasi-mental fact which corresponds and which goes along with the motion of every particle of matter is of such inconceivable simplicity, as compared with our own mental fact, with our consciousness, as the motion of a molecule of matter is of inconceivable simplicity when compared with the motion in our brain.

"This doctrine is not merely a speculation, but is a result to which all the greatest minds that have studied this question in the right way have gradually been approximating for a long time."

We should have thought that the consciousness of inorganic matter was one of those things which the author would have put behind the moon—as one which may or may not be, but which it is, can never be known. Moreover, the complexity of the brain may be a measure of its consciousness, but is no reason for it. The kidney is also complex and so is Babbage's machine; but the complexity of the brain is important, because the organ has been developed for consciousness, and even in comparing acknowledged examples of consciousness in animals, there must be a vast interval between that of an animal possessing an organ developed for it and of an animal in which there is no centre of sensibility. Elsewhere, Clifford says in a letter,

"I am a dogmatic nihilist, and shall say the brain is conscious, if I like. Only I do not say it in the same sense as that in which I say that *I* am conscious. It seems to me that not even Vogt, however you fix it, can talk about matter for scientific purposes except as a phenomenon; that in saying the brain is conscious—or, better, that *you* are conscious, I only affirm a correlation of two phenomena, and am as ideal as I can be."

This combination of metaphysical idealism with scientific belief and method, not peculiar to Clifford, is, however, a somewhat new philosophical position, far more difficult to assail than that of pure realistic science. Clifford had worked

out his metaphysics a good deal for himself, he had read little on the subject, his favourite authors, however, being Spinoza and Berkeley.<sup>1</sup> Had he lived, he would, we think, have been likely to have left a strong and deep mark upon the metaphysical speculations of his time, from his habit of ever attempting to get at the central truths of speculative opinion from new points of view. The volumes before us are full of instruction and interest.

JOHN CHARLES BUCKNILL.

<sup>1</sup> On this question of consciousness we remember a paper of Clifford's which we do not find in the present collection, and in which he drew an important distinction between the ordinary objects of consciousness, and our consciousness of other consciousness, which indeed is proved to us objectively, but which we do not recognise as in itself an object.

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## Clinical Cases.

### CASE OF PURULENT CEREBRAL AND SPINAL MENINGITIS—NO SYMPTOMS—SUDDEN DEATH BY COMA.

UNDER THE CARE OF JOSEPH W. HUNT, M.D. LOND.,

*Wolverhampton and Staffordshire General Hospital.*

MARTHA V., æt. 60, married, came to the out-patient room Feb. 27th, complaining of dyspnœa and pain in the back.

As she came from a great distance, she was at her own request admitted as an in-patient, though scarcely considered fit for admission.

On examination in the wards she was found to be rather badly nourished. Expression careworn. Capillaries in face well marked. No general or local œdema. No cutaneous eruption. Temperature, taken in armpit, normal (at night). She complained of much pain in the lumbar region, which was greatly increased by walking, coughing, or by raising herself up after stooping. It had been present, with only short intervals, for nearly fourteen years, and never extended into any of the limbs. There was no spinal tenderness beyond a little undefined uneasiness on firm pressure being made over the base of the sacrum. No headache. Sight and hearing good. No ophthalmoscopic examination was made. Intellect very good. No abnormalities with regard to either motion or sensation. Slept well. Considerable dyspnœa on exertion, but only a little cough and very scanty expectoration. Physical signs of emphysema well marked. Cardiac impulse and sounds weak, and some cardiac dilatation. Tongue clean. No vomiting. A tendency to constipation and some dyspepsia. Urine 1016: acid, rather turbid, with a deposit of phosphates on heating, abundant in quantity; no albumen.

The diagnosis of muscular rheumatism and emphysema was made, and patient was ordered potass. bicarb. gr. xv., inf.

calumbæ 3j ter die, and the bowels were kept regularly open with liquorice powder.

Under this treatment the patient improved rapidly. The muscular pain almost entirely left her. She was able to get about the ward well, and expressed herself very grateful for what had been done. No fresh symptoms declared themselves. On March 7th, eight days after admission, she breakfasted at 6 A.M. as usual, and told the nurse how much better she was. At 8 P.M. she was observed by the nurse to be to all appearance asleep; but on a closer examination the nurse was alarmed, and Dr. Russell, the House Physician, was summoned. He saw her at once, and made the following notes: Patient lying on her back, and breathing heavily and noisily; eyes closed, but on being sharply addressed they were opened, and patient would try to speak, but never uttered more than "Ah!" Pupils reacted to light: about normal in size, the left being a little more contracted than the right. No strabismus, and no cranial paralysis. She swallowed her drink well. The tongue was not protruded, though it was occasionally seen to move about in the mouth. No paralysis of limbs, which were frequently moved in a restless manner, and almost invariably whenever they were pricked. There was no exaltation of sensibility, but rather the reverse, though patient could feel a prick on any part of the body. The legs were bent up in bed, especially the left; in this limb the leg being strongly flexed on the thigh, and the thigh on the abdomen, so much so that before death the knee almost touched the abdominal walls, but at times she fully extended the limbs. Face rather cyanotic. Urine passed into bed. Pulse 120, irregular in force and rhythm, and intermitting. Temperature carefully taken by Dr. Russell. Right axilla 103°. Left axilla (same thermometer) 102·4°.

1 P.M.—No marked change. Temp. at right axilla 101·7°; left, 100·6°. Over the frontal regions the temperature was about the same on the two sides (101·5°). She did not recognise her friends who visited her at about this time. There was slight retching, but no vomiting. Once or twice, for about five minutes, nurse noticed some twitching of the lower lip, most marked on the left side.

5 P.M.—Temp. right axilla, 101·8°, left, 101°. Eyes kept open, instead of closed.

8 P.M.—Pulse 132, much weaker, and very irregular; no evidence of paralysis anywhere. When the limbs are raised they fall gently, and not as a heavy weight.

She died comatose at 11 A.M., 27 hours after onset of coma. Post-mortem made and reported by Dr. Russell 24 hours after death. Rigor mortis and hypostasis well marked. Calvarium

removed in usual way. Exterior of dura mater normal, but on cutting through it a considerable amount of pus flowed out; and on removing the dura mater the surface of the brain was seen to be covered with pus in its greater part. It was most plentiful over the posterior portion of the frontal convolutions, the ascending frontal and the ascending parietal, and the 1st, 2nd, and 3rd parietal convolutions; and at those situations too it was removed with greater difficulty when a stream of water was poured on the surface. There was no marked difference in the amount of pus between the two sides. The anterior half of the frontal lobes and the occipital lobes were pretty clear of pus. There was pus too at the base of the brain, but none over the cerebellum nor about the medulla. The membranes were much injected. Vessels normal, considering the age of the patient, and there was no thrombus or embolon in the larger vessels. Sinuses normal. There was no disease of the bone found, though it was carefully looked for, and each internal ear was healthy. No appearance of a fracture or fissuring in the skull. On section of the brain, the puncta cruenta were seen to be more than ordinarily numerous and well marked. The white substance was of a brownish-pink colour throughout, but both it and the cortical substance appeared to be of about normal consistence. No lesion of any kind was seen in any portion of the brain. On reflecting the dura mater of the cord it was seen to be bathed in pus throughout, with the exception of the upper three inches, and equally so in front and behind. The dura mater was normal in appearance, but the other membranes were markedly injected. The substance of the cord was in every way normal to the naked eye. The vertebræ were healthy. Heart weighed 10 ounces, right side dilated, substance soft. Lungs emphysematous. Spleen, 6 ounces, very pulpy. Kidneys,  $4\frac{1}{2}$  and 5 ounces. The capsules of both were adherent in places, and markedly so in one kidney, so that the renal substance was much lacerated in removing the capsule. Both contained cysts in their cortex. There was some retroperitoneal hæmorrhage in the neighbourhood of the left kidney, but no lesion in the kidney or ribs to account for it. No abscesses or emboli found in any portion of the body.

*Remarks.*—The history of this case presents several points of interest. Non-traumatic meningitis is by no means a disease of frequent occurrence in advanced life, but the opposite. True, several instances are to be found scattered throughout various publications, especially those of France and Germany, and Schönlein (as quoted by Day) even goes so far as to write that meningitis is especially a disease of old age; but, on the

other hand, those investigators who have laboured at such institutions as the Bicêtre and Salpêtrière—Durand Fardel, for instance—mention the rarity of this disease, and more especially of the purulent form. The cause of the affection in this particular case is involved in obscurity. No history of injury was obtained, and the surrounding bones and the internal ear were healthy. As far as was known, she was a temperate woman, and the post-mortem condition confirmed this—and even if it were otherwise, it is doubtful whether alcoholism is anything more than a predisposing cause. It is possible that the meningitis may have been associated with the cirrhotic changes found after death, though during life there were no indications of renal disease. Dr. Maclachlan writes in his work on the ‘Diseases of Advanced Life:’ “Almost every case occurring in the old may be traced to one of three sources, gout, rheumatism, or albuminuria.” The history of the case is strongly against the idea of its being epidemic cerebro-spinal meningitis; moreover, this latter disease is very rare in those over 50. Except in very severe epidemics, it is never so apoplectic in its course, and, post mortem, the posterior portion of the dura mater of the cord is always found to bear the brunt of the affection.

That this was a chronic affection with latent symptoms is controverted by the normal thickness of the membranes and the naked-eye appearance of the inflammatory products, though of course it is most probable that the disease commenced some short time before she became comatose. The fact that a patient apparently in good health should suddenly become comatose and die in 27 hours, and nothing but purulent meningitis be found post mortem, must considerably complicate the diagnosis of the causes of apoplectic seizures. Dr. Maclachlan, in his work already referred to, mentions cases of meningitis of an “apoplectic character,” and adds that there is generally a history of intemperance. A case is recorded in the ‘Medical Times and Gazette’ (Nov. 2, 1867), under the care of Dr. Hughlings-Jackson. The patient was picked up in the streets, and, with help, walked to the receiving-room of the hospital, where he became comatose with stertorous breathing, and died 26 hours afterwards. The lesion found after death was purulent meningitis. In his case he had complained of considerable headache a few days previously.

## CLINICAL CASES OF HERNIA CEREBRI.

BY JOHN DUNCAN, M.A., M.D., F.R.C.S., ETC.

*Surgeon to the Royal Infirmary, Edinburgh.*

THE following cases at the time of their occurrence appeared to be merely ordinary examples of the curious variety of effect produced by similar injuries of the brain. Viewed now in the light of recent researches, and when so much that is important and valuable is being made out in reference to localisation of function in the brain, they assume a somewhat different aspect, and at least seem to me worthy of record.

On the 22nd of March, 1872, Stephen Maclachlan, a sailor, twenty-four years of age, was entering a boat to be rowed to his ship, then lying off a Spanish port, when he was struck on the head by a stone thrown at him from above. He was carried completely insensible to a neighbouring hospital, and remained so for several hours. Of his stay there we know nothing farther than that he was copiously and repeatedly leeches and bled, and that a companion, who visited him during the third week, observed a considerable swelling on his head at the seat of injury.

On leaving, after four weeks, he was taken home to Grangemouth by his friends, and thence transferred to the Royal Infirmary on the 11th of May.

On admission the patient was found to be a well-built powerful-looking man, but anæmic.

Over the left parietal bone, opposite the centre of the sagittal suture, was a large hernia cerebri, whose inner margin reached the middle line. Its antero-posterior diameter was 3 inches, its transverse 2 inches, and it was  $1\frac{1}{2}$  inch in depth. Its surface was irregularly nodulated, having a yellow sloughy aspect, but with here and there a few pale granulations. It overhung the surrounding skin. The hernia itself was quite insensible to touch, but the scalp under and about it was somewhat swollen and tender. The tumour pulsed strongly.

The following notes of his condition were made :—

The patient has lost the power of speech. He makes no attempt to speak, and if asked to repeat a word, he can only very imperfectly imitate the sound of the most prominent syllable. He cannot write the shortest word, and makes curiously unpronounceable combinations of things that look like letters.

His intelligence, however, appears good. He can make signs expressive of simple wants, and by nodding or shaking his head he answers with correctness questions that require considerable reasoning power. If spoken to quickly, he requires a little time to understand, and sometimes the question has to be repeated.

He has completely lost the power of moving his right arm and leg. The right side of the face is paralysed. There is not much drawing to the left, but a distinct absence of expression when he laughs, and he smokes with the pipe in the left side of the mouth. The right eyelid droops considerably, and he cannot shut it completely unless he also closes the left. The tongue is protruded nearly straight. The bowels and bladder act perfectly. He has twitching of the right arm and leg, principally at night.

Sensation is normal on the left side. On the right side a light touch is not felt at all. There is a degree of pressure which gives him sensation, but without enabling him to say whether one or two points touch him. On very firm pressure with the compasses he distinguishes points almost although not quite as well on the right side as on the left. Pressure, rubbing, or other rough movement of any portion of the right side gives considerable pain. This is very marked in the arm and thorax, less so in the leg. He feels acutely if the sole of the right foot be tickled, but the reflex movement is only a slight twitching of the toes.

The right pupil is dilated, reacts slowly to light, and its vision is slightly impaired. (The ophthalmic report was unfortunately not preserved.)

He cannot hear the watch with the right ear unless it be applied to the auricle.

On the left side sight and hearing are normal.

No distinction can be made out between the two sides as to smell and taste.

All other organs seem to be healthy and working well. The appetite is good, the urine normal. Pulse 84, strong and regular; temperature 99·8.

Absolute rest was enjoined, and the hernia was dressed with carbolised oil.

The patient progressed favourably up to the 29th of May.

A certain amount of power returned to the limbs, especially to the leg, although a progressive diminution in the bulk of the paralysed members took place, in spite of the regular use of the induced current. The hernia looked more healthy, had slightly diminished in size, and a narrow margin of growing cicatrice was visible at the circumference. The pulse varied from 80 to 90, and the temperature from  $99^{\circ}$  to  $100^{\circ}5$ .

On the 29th, however, he did not sleep well, and next morning the temperature had risen to  $102^{\circ}$  in the right axilla,  $101^{\circ}5$  in the left. On gently pressing the tumour a drop or two of pus could be made to exude from a very minute opening on the right side close to the base. Into this a probe could be introduced nearly one inch.

Next day there was a rapid rise of temperature to  $108^{\circ}$ , with much headache. The pulse fell to 64. The opening was enlarged, and a small quantity of pus evacuated.

No improvement followed, and next day, June 3rd, as he was becoming comatose, the hernia was sliced off, and an abscess cavity, the size of a walnut, was disclosed.

He went, however, from bad to worse. On the 4th he was completely comatose. The right pupil became enormously dilated, and there was most profuse perspiration of the whole head and face.

On the 5th a copious flow of what seemed diffluent brain-substance took place, and he died on the morning of the 6th of June.

During the last few days striking variations occurred in the temperature. At one time it would be  $107^{\circ}$  or  $108^{\circ}$ , and a few hours afterwards normal. In the right axilla it was generally a degree higher than in the left. The pulse remained under 70 till the evening before his death, when it rose suddenly to 150.

A post-mortem examination was made next day. On removing what remained of the hernia, an oval opening in the bone was disclosed, an inch and a quarter long by three-quarters of an inch broad. Its inner margin was one quarter of an inch from the suture, and on both sides the bone was considerably depressed. The anterior margin of the aperture covered the upper part of the ascending frontal convolution, the rest passed backwards over the ascending parietal convolution and the superior parietal lobe. The interior of the parietal lobe was occupied by an abscess cavity containing diffluent brain-tissue and pus. The upper part of the ascending frontal convolution was involved in the abscess, which otherwise corresponded with great accuracy to the parietal lobe. The rest of the brain appeared healthy.

On the 6th of June, 1869, David Fergusson, æt. 13, was kicked on the forehead by a pony, while playing with some companions in the stable. I saw him, along with the late Dr. Andrew Inglis, shortly after the accident. I found that the left temple was completely smashed. There was a circular orifice about the size of a penny in the bone, its upper margin just touching the hairy scalp. A small quantity of brain-substance lay among clotted blood on his cheek. In the wound lay a mass of blood and debris, and a piece of bone driven in edgeways for more than an inch into the brain. The pulse was 90, the temperature normal. He was able perfectly to describe the mode in which the accident occurred, and I learned that he had not lost consciousness for a moment. There was in short absolutely no apparent disturbance of function in any portion of the body. I removed the loose portions of bone, elevated a piece which was slightly depressed, and dressed the wound with a single layer of lint moistened with water.

It would be tedious to transcribe from my notes the uneventful history of the case. Let me merely say once for all that throughout there was no appreciable alteration in any function, bodily or mental. The comparative power and sensibility of the two sides of the body were carefully and repeatedly tested, and found normal. He was in all respects a quick intelligent boy, of a bright and pleasant disposition, and his father and companions frequently told me that they were unable to detect the slightest change in his mental capacity or disposition.

On the eleventh day after the accident a hernia cerebri began to develop. In ten days it attained the size of a small orange. From that time it remained stationary for four months, being left entirely to nature, except in so far as a most rigid cleanliness was observed. At the end of that time it began to dwindle, the cicatrice drew in from the margins, and six months after the injury a level pulsating scar was all that remained.

Shortly afterwards I lost sight of my patient.

After three years one of the resident physicians in the medical department of the Infirmary informed me that Fergusson was then dying of phthisis, a disease which had killed his mother. I saw him, and was present at the autopsy, and carefully examined the injured portion of the head.

An almost circular opening, one inch in diameter, with thin and smooth edges, occupied the left side of the frontal bone. From this, as a base, an irregular cone of cicatricial tissue extended into the brain for three-quarters of an inch. This cone occupied the centre of the second, and slightly the contiguous part of the first frontal convolution.



These cases present several points of interest, both in their clinical and pathological aspects.

The amount of brain-tissue directly injured was at least as great in the kick from the horse as in the blow from the stone, yet in the effect produced the contrast is very striking. The initial period of unconsciousness in Maclachlan's case was prolonged and complete; in Fergusson's there was none whatever. That is a symptom which is always difficult of explanation. Had the respective ages of the patients any relation to the early phenomena of concussion? Had the direction of the blow an influence in transmitting the violence more directly to the basal ganglia in the one case than the other? Was the very force and suddenness of the kick an element in diminishing shock? or was there irritation in Maclachlan's case from the depressed bone? To this last cause I attribute the fatal result. In Fergusson's case the depressed and fractured bone was at once elevated and removed; in Maclachlan's the *sectio cadaveris* showed that this had not been done. When he was brought to the Infirmary, it was then too late even to ascertain that such depression existed.

Doubtless the widely different symptoms of interference with nerve function depended upon the portion of the brain injured. They bear out to a certain extent the conclusions which have been drawn from recent researches, in so far as the great regions, frontal and parietal, are concerned. But they present striking anomalies. It is a remarkable fact, whether we are to explain it by duplicate action, substitution, or insignificance of function, that a considerable portion of the second frontal convolution may be destroyed without any palpable sign of its absence having been manifested during a period of three years. And not less remarkable is it in Maclachlan's case, considering the mass of evidence that has now been accumulated with reference to the localisation of function in the third frontal convolution, that to the naked eye that portion of the brain was absolutely uninjured. If, as in the case lately published by my friend Dr. Cunningham, we may have Broca's convolution destroyed without aphasia being produced; and if, on the other hand, as in this case, we may have the most complete aphasia while it remains apparently unaffected; it becomes exceedingly difficult to reconcile the ascertained facts. That such a reconciliation will be made out no one of course can doubt; but whether it is to be sought for in the double nature of the brain for the one case, and in compression or reflex action for the other, I do not venture even to discuss.

## CASE OF ACUTE ASCENDING PARALYSIS.

BY ARTHUR W. FOX, M.B.

*Physician to the Eastern Dispensary, and Bellott's Mineral-Water Hospital, Bath.*

ON March 21st, 1879, I was asked by Dr. F. Berry, the resident Medical Officer to the Eastern Dispensary, to see with him the following case:—

F., aged 26 years, a tall girl of brunette complexion, had led the life of a prostitute for the last seven years, and had been a great drinker of beer and gin; had never gone to bed sober for nearly two years; often exposed to damp and cold; five or six years ago had syphilis, history of sore, followed by eruption and sore throat. Ten months ago had a fit; was unconscious, was convulsed and foamed at the mouth. For several months she had complained of great languor and loss of appetite, and during the last six weeks those about her noticed that when she sat down in a chair she had to catch hold of something to pull herself up again. She had not menstruated for nearly two months. A week ago her present symptoms began with pains in her legs and loins; this was succeeded by incontinence of urine, and on March 30th by loss of power in her legs.

*Present state.*—In bed, lying on her back; pupils equal, act to light; face looked swollen, pale, and shining; tongue-edges and tip red, dorsum covered by a dry brown fur: some loss of power in both arms, more so on left side; there is complete loss of power in both legs; hyperæsthesia and hyperalgesia are also present in lower limbs—of the latter a high grade, as touching her in the gentlest manner makes her scream out, also the same on moving her legs and tickling the soles of her feet; tickling her left sole produces a slight flicker in quadriceps extensor of right thigh; the tendon reflexes appear to be abolished. She complained of pain in her back and legs of a dull heavy character. On inner aspect of both thighs are some copper-coloured spots, evidently an old syphilitic eruption; passes water involuntarily in full stream; bowels not open for four days; pulse 160; respirations 30; temperature 98.

*March 22nd.*—More motor paralysis of arms, attended with hyperæsthesia and hyperalgesia: the hyperæsthesia not so marked in legs; tickling right sole gives rise to slight fibrillary tremor in both thighs, more so on right side; speech affected; pulse 160; respirations 30; temperature 98.

*March 23rd.*—No water passed since last night, but no dulness above pubes, abdomen tympanitic; cries out when she is touched or percussed; bowels not open; pupils more dilated; tongue protruded as far as lip; speech so indistinct as to render it almost impossible to understand her; semi-delirious; no difficulty in swallowing; arms more paralysed; pulse 160; respirations 30; temperature 101.

*March 24th.*—Rambling delirium during night; speech much the same as yesterday, only feebler; sordes on lips and teeth; tongue covered by a dark brown dry fur, can only protrude the tip as far as the edge of lower lip; no difficulty in swallowing; condition of limbs the same as yesterday; perspires profusely; left pupil dilated oscillating; passed water yesterday evening. Specific gravity 1030, acid, ammoniacal; albumen one-sixth, brick-coloured urates; no action of bowels; pulse 160; respirations 24; temperature 99·6.

*March 25th.*—Dozing all the morning; face flushed of dusky hue; pupils equal, act better to light; bed-sore forming on left buttock; no other change in her symptoms; pulse 152; respirations 32; temperature 99·8.

*March 26th.*—In much the same condition; almost complete motor paralysis of both arms, can slightly move right wrist; motor paralysis of legs the same, also the hyperalgesia of arms, legs, and body, but there is an amount of anæsthesia; upon pinching her legs there is evidently slow conduction of the impression, as there is an interval of three seconds before she cries out; reflex excitability completely extinguished; there is no apparent atrophy of the muscles of the limbs; speech very feeble and indistinct. Her mother can understand that she complains of feeling numb all over. When I asked her if this was the case, she said "Yes." No stiffness of neck; can move her head in various directions; no action of bowels; pulse 148; respirations 30; temperature 100·4.

*March 27th.*—Much the same; pulse 152; respirations 32; temperature 100·4. Between 7.30 and 10.30 yesterday evening her skin was covered with a profuse perspiration, which Dr. Berry found to be of an alkaline reaction.

*March 28th.*—Pulse 160; respiration 32, heaving; temperature 100·2. Can speak only in a whisper; face dusky, of semi-livid tinge; left pupil larger than right. No other changes in her symptoms.

*March 29th.*—She died at 2 A.M.

The autopsy was made in my presence by my friend and colleague Mr. F. K. Green, assisted by Dr. Berry, on March 30th, thirty-seven hours after death.

Body well nourished; rigor mortis well marked, bed-sore on each gluteal region. The spinal cord and membranes were removed, the latter being left intact: there was no abnormal appearance in the spinal canal. Membranes of brain normal; no appearance of disease; vessels at base of brain substance appeared normal; basal ganglia, corpus striatum, and optic thalamus on each side contained some small punctiform hæmorrhages. Thorax, pericardium normal. Heart of normal size, walls soft, the finger could easily be pushed through the left ventricle; valves and orifice normal. Aorta and pulmonary artery normal. Lungs—lower lobes gorged, upper lobe of left contained a hard mass sharply defined from the surrounding lung structure; a few smaller nodules like that in left lung were found in lower portion of upper lobe on right side. Abdominal wall contained a layer of fat an inch thick. Upon the omentum and upon the abdominal wall near the spleen, and on the under-surface of diaphragm were situated numerous small semi-transparent bodies (tubercle). Liver large and fatty, a small yellowish-red nodule was situated on anterior surface of right lobe; gall-bladder, empty of bile, contained a few gritty particles. Spleen small, diffluent: pancreas normal. Kidneys of normal size, lobulated, capsules peeled well; upon section, structure of kidneys appeared congested, but otherwise normal. I submitted to my friend Dr. Shingleton Smith, for examination, the cord and its membranes, the base of brain, the mass in left lung, and the nodule on the liver. The following is the report that he has kindly furnished me with:—

“The nodule found on the surface of the liver has the structure of a small gumma which has undergone some degeneration; it appears to have grown in the wall of, and around, a small artery. You will notice a central aperture in each of the sections, but no normal arterial texture remains. The connective tissue element at the surface of liver in the neighbourhood of this nodule is in excess, but the liver cells are in a state of advanced fatty degeneration.

“The solid mass found in the lung has a fibrous structure, the air cells are entirely obliterated, little normal epithelium can be made out. Such a dense fibrillated mass can result from little else than a gummy tumour, which has been undergoing organisation for a considerable period. There is no appearance of any tuberculisation of lung, and the mass is too well defined and limited to be of inflammatory origin.

“The spinal cord did not present any abnormal naked-eye

appearance. The vessels of the pia mater were full, and there seemed to be an excess of connective tissue binding together the spinal nerves in the cauda equina. Numerous small nodules noticed on either side in the connective tissue mass were found to be healthy spinal ganglia on the roots of the nerves.

"Sections of the cord in the cervical region seem to have some increase in the connective tissue elements. The grey matter took the staining fluid more readily than usual, and some of the cells have a contracted appearance. In the lumbar region no special appearances were observed. Sections through the medulla oblongata stained very readily and deeply after short immersion in log-wood fluid, but no other appearance deserves comment. At present I can only say that the appearances are consistent with a diffuse syphilitic inflammation. No definite new growth exists in any portion of the nervous system, and no masses of leucocytes are seen in the sections; the change, if there is any, is a very diffuse one. In the absence of any other morbid change, the intense coloration is an indication of some minute and diffuse protoplasmic change, such as would exist in the early stage of diffuse myelitis."

*Remarks.*—Taking into consideration the very slight changes found in the cord by Dr. Smith, the case resembles in many respects those described by Landry, Kussmaul, Westphal, and others, as acute ascending paralysis. The diagnosis during life lay between acute ascending paralysis of syphilitic origin, such as Heubner describes, acute central myelitis and acute anterior polio-myelitis. The latter affection appeared to be excluded, as, according to Erb, it never attacks the medulla oblongata, whereas in the case of this woman she was unable to protrude the tip of her tongue beyond the lip, and her speech was markedly affected for at least seven days before her death. In addition to this absence of bulbar trouble in acute polio-myelitis anterior, there is neither a tendency to formation of acute bed-sore, nor are the sphincters affected to any great extent; besides, the sensibility of the skin remains intact, the disease is also ushered in with pyrexia, and is followed by rapid atrophy of the muscles of the paralysed limbs, which was certainly not appreciably present in the case of this patient. There is also a rapid loss of the faradic excitability; the latter unfortunately was not tested, as we have at present no reliable electrical apparatus at the Dispensary. On the other hand, she had certain symptoms similar to those seen in acute central myelitis, viz. the rapidly increasing failure of the reflex excitability, the formation of acute bed-sore, the

paralysis of the sphincters and the disturbance of sensibility : in this patient's case the hyperæsthesia at the commencement, and the hyperalgesia throughout, were so marked as to suggest the notion that there was probably some meningeal complication. But Erb states that in single cases of acute ascending paralysis a high grade of hyperæsthesia (hyperalgesia) of the skin has been observed. Against the diagnosis of acute central myelitis was the entire absence throughout of any convulsive movement. Erb, however, remarks that a positive diagnosis between these two diseases in many cases can only be made with the help of the autopsy, but he afterwards qualifies this statement by saying that in acute ascending paralysis no anomaly of electrical excitability worth mentioning has been found even after the disease has existed for a number of weeks, a point which constitutes a marked distinction between this form of disease and all progressive paralysis caused by gross anatomical changes within the cord, such as central myelitis and polio-myelitis anterior.

Whether this patient's case was one of genuine acute ascending paralysis, or a spinal form of syphilis which ran the course of acute ascending paralysis, like those alluded to by Heubner, it seems impossible to determine, as the symptoms noted during life, and the absence of any marked anatomical lesion in the nervous system, are common to both diseases; the only evidence in favour of the latter being the previous history of syphilis, and the appearances found in the liver and the lung.

*[Read before the Bath Pathological and Clinical Society.]*

## PUNCTURE OF LENS. OPHTHALMITIS. BRAIN DISEASE. DEATH.

BY T. PRIDGIN TEALE, M.A., F.R.C.S.

*Surgeon to the General Infirmary at Leeds.*

THE following case, no less interesting than it proved to myself to be distressing, seems worthy of being placed on record.

In May 1867 I was consulted by Mr. J. P. I., aged 45, a celebrated Yorkshire tenor, about an increasing defect in his sight, which was seriously interfering with his profession as a singer and teacher of singing. He was myopic, and had half-formed cataracts in both eyes.

In order to economise his time I decided to accelerate the formation of the cataract in one eye, so that it might be ready for extraction during the vacation which would commence in three weeks. With this object, I slightly ruptured with two needles the anterior capsule of the left lens, in which the nuclear opacity was most advanced, rendering the eye useless. The rupture of the capsule was but slight, as it was my intention to produce slow formation of opacity of the cortical layers, with as little excitement as possible, so that he might at the same time continue to give music lessons during the three weeks which would elapse before the holidays. I name this to show how slight a rupture of the capsule it was my aim to make.

*May 24th.*—Puncture of lens by needles. During the afternoon the eye was more painful than is usual after such a slight operation, and the conjunctival vessels were injected.

*May 25th.*—Great pain. No appearance of opacity nor swelling of the lens. *The punctures in the cornea look opaque.* Some sickness during the day.

*May 26th.*—In the night intense vomiting, with agonising pain in the eye. During an attack of vomiting "he felt the eye go blind." At 10 A.M. I found the eye hard (Tension  $+1\frac{1}{2}$ ), the cornea dull, with opaque infiltrations (purulent) at the margin, and the pain intense. He considered the sickness

as one of his bilious attacks, to which he had been liable in spring and summer.

*May 28th.*—Cornea has sloughed, and the lens has escaped.

For a while I hoped that the remnant of the eye would become quiescent, and that we had seen the worst in the loss of sight. Pain, however, increased, and his distress was so great that I decided to extirpate the eye.

*June 4th.*—The eye was extirpated.

*June 5th.*—Much swelling of the lids and orbital tissues.

*June 7th.*—Swelling almost gone. He sat up, and went downstairs.

*June 8th.*—Early in the morning, after a restless night, with chilliness but no rigor, he felt an intensely distressing fulness and throbbing at the back of the head on the left side, and had become aphasic, being hardly able to articulate or remember words, using wrong words, and mixing words together.

*June 9th.*—Improving; able to speak with tolerable distinctness.

*June 10th.*—The same. Pain at the side of the head. Often looks flushed. Left hand weak.

*June 12th.*—A convulsion.

*June 13th.*—Several convulsions.

*June 14th.*—Convulsion early in the morning; none after. He then gradually became less able to speak, more dull, but still conscious, taking very little food, and often flushed.

*June 18th.*—He died, with convulsive twitches of the left side, the right side being apparently paralysed, and the face slightly drawn to the left.

*Post-mortem.*—Dura mater was firmly adherent at several parts of the calvarium. There was very little fluid in the arachnoid cavity, and there were no adhesions of opposed surfaces of the arachnoid. There was no appearance of disease of the optic nerves, no pus about the cavernous sinus, nor at the base of the brain.

On the upper surface of the left hemisphere the arachnoid was thickened, and the subarachnoid space was filled with greenish firm lymph which looked like pus, but did not ooze out on section, and could not be displaced by pressure. Near the upper surface of the left hemisphere, rather behind the middle, there was a mass of disease involving the grey matter and centrum ovale, not invading the wall of the lateral ventricle, but situated above its roof. Both corpora striata and optic thalami were cut into without finding disease. The diseased mass consisted of a centre of the size of a small walnut of uniform chocolate tint, and in firmness like healthy brain, surrounded by a rather softer portion of brain studded with small round



black clots, the largest of the size of a large pea. The discovery of this disease, some of which clearly had not wholly arisen subsequently to the operation, led me to make inquiries, whereby the following facts were drawn out, of which I had never received the slightest hint.

About two years before this disastrous operation he jumped down from a coach with which the horses were running away, and fell, his head forcibly striking the ground. He then got up, overtook the coach, assisted with the horses, and became unconscious for several hours. After the accident he had on two or three occasions fallen from sudden unconsciousness.

The notes of the appearances in the brain were not made at the time of the post-mortem examination, but from memory shortly after.

## CASE OF PSEUDO-HYPERTROPHIC SPINAL PARALYSIS OCCURRING IN AN ADULT.

BY A. HUGHES BENNETT, M.D.

THE following case is worthy of record as being an example of a disease by no means common amongst children, and which is extremely rare in the adult.

John Mill, aged 26, a hawker. The patient states that until eighteen months ago he was a perfectly healthy man. Prior to this period he had passed his life much as other persons in his class of society do. He had been to school as a boy, and at the age of seventeen he had become a gardener, which occupation he continued to practise till four years ago, when he changed his mode of living to that of a hawker. This calling he has since continued to favour with his patronage, except when occasionally required by Her Majesty's Government to pick oakum in gaol. In all these occupations, whether voluntary or forced, he never had any excessive amount of labour, and he never had any difficulty in doing an ordinary day's work.

About eighteen months ago he became afflicted with uneasy sensations in all the muscles of his body, chiefly those of the thighs, with stiffness in walking, and with rheumatic-like pains. It was then for the first time discovered that many of the muscles were enlarged. The patient states that he had not noticed this before, and was unable to say when the hypertrophy commenced. Distinct weakness of the legs soon supervened, which so increased as considerably to affect his gait. Three months later the arms became so feeble that he was unable to perform the duties to which he had been accustomed, and the limbs were found increased in size. Since then his condition has remained the same till he came under observation.

*Present Condition.*—A healthy-looking man. Height, 5 ft. 6½ inches; weight, 156 lbs. *Attitude.*—While sitting, finds it wearisome to remain upright, and usually prefers to recline.

While standing, places feet and legs widely apart, the pelvis is thrown forward, and the shoulders backward, so that the dorsal convexity overhangs the nates. *Muscular Conformation.*—On inspection, most of the prominent muscles of the body are found greatly enlarged, especially those of the neck, back, abdomen, thighs, and calves. The temporal and masseter muscles on both sides are enlarged, firm, and bulge outwards, so as to give a peculiar expression to the individual. The muscles of the neck are all remarkably hypertrophied, giving the patient a bull-necked appearance. The neck about its middle measures in circumference 15 inches. The tongue is larger than natural. The muscles of the chest and back are well formed, but not specially enlarged. Girth of chest below axilla is  $36\frac{1}{2}$  inches, at level of nipples 37 inches. The deltoids are well developed, so also are the muscles of the upper and fore arms, neither of which are larger than those of a healthy working man. The measurements are :

	Right.	Left.
Upper arm at axilla, when extended . . .	$10\frac{1}{2}$ in. ..	$10\frac{3}{4}$ in.
"    when flexed . . .	11 ..	$10\frac{3}{4}$
Upper arm at middle, when extended . . .	$10\frac{1}{2}$ ..	$10\frac{3}{4}$
"    when flexed . . .	11 ..	$11\frac{1}{4}$
Upper arm above elbow, when extended . .	$8\frac{1}{2}$ in. ..	$8\frac{1}{2}$ in.
"    when flexed . . .	$9\frac{1}{4}$ ..	$9\frac{1}{4}$
Fore arm below elbow . . . . .	10 ..	10
"    at middle . . . . .	$8\frac{1}{2}$ ..	9
"    above wrist . . . . .	$6\frac{1}{2}$ ..	$6\frac{1}{2}$

The muscles of the abdomen and lumbar regions are considerably enlarged and prominent, so as in the latter to leave a deep sulcus in the spinal line. The girth at level of umbilicus is 33 inches. The glutei on both sides are very greatly enlarged, the circumference, when both legs are together, being  $37\frac{1}{2}$  inches. The muscles of both thighs also are very much hypertrophied, the measurements being :

	Right.	Left.
Thigh at level of great trochanter . . .	$22\frac{1}{4}$ in. ..	$22\frac{1}{4}$ in.
"    at middle . . . . .	22 ..	22
"    above knee . . . . .	19 ..	19

The muscles of the calf of the leg on both sides are very much enlarged, the measurements being :

	Right.	Left.
Calf at thickest part, flexed . . . . .	$15\frac{3}{4}$ in. ..	$15\frac{1}{4}$ in.
"    extended . . . . .	$15\frac{1}{4}$ ..	$14\frac{3}{4}$
Above ankle joint. . . . .	$7\frac{1}{2}$ ..	$7\frac{1}{2}$

All the other muscles of the body, except those especially described as enlarged, seemed to be abnormally small, the two being in striking contrast. The hypertrophied muscles were firm, dense, and hard to the feel, and this was further increased

when attempts at flexion were made. The faradic and galvanic currents being applied, the muscles contracted feebly, but far short of normal action. An effort was made to induce the patient to permit a portion of his muscle to be submitted to microscopical examination, but his thirst for scientific research was so feeble as to be overcome by the fear of the small operation necessary for the purpose. *Sensibility*.—Functions of brain normal, with exception of occasional pain in the vertex. Has a feeling of formication, and a pricking sensation in the thighs. He has also a peculiar twitching feeling in the muscles. Has no dorsal or other pain. Sensibility in other respects normal. *Reflex Motion*.—Normal. *Voluntary Motion*.—In almost exact proportion to the increased size of the muscles is their strength and power of movement impaired. The movements of the tongue are sluggish, and can only be performed slowly and deliberately. The patient has difficulty in commencing to speak, but having once begun can articulate fairly well. The muscles of the jaws and face act slowly and feebly, so also do those of the head and neck. Movements of the chest appear normal. The upper extremities can be moved freely in all directions, and there is everywhere perfect power of co-ordination. Considering the size of the muscles, there is great want of power, the movements of the limbs being slow, feeble, but steady. The grasp of the hand is weak, the right the most so, although formerly the patient was not left-handed. A pin can be taken up accurately, but cannot be used neatly. The lower extremities are both extremely weak. The patient walks with much difficulty, with a feeble, waddling gait, swaying from side to side, and with his legs wide apart. He finds it almost impossible to go up-stairs; rises from the sitting position with much difficulty, having to assist himself with his arms. When he lies on his back he can move his legs in all directions, but slowly and imperfectly. *The Special Senses* were normal, with the exception of the right eye, the sight of which was greatly diminished, and the condition of which was as follows:  $S = \frac{1}{7} \frac{o}{o}$ ; the retinal vessels small; pigment spots in neighbourhood of optic disc; otherwise normal. The other systems of the body were normal.

*Remarks*.—Few observations are necessary in a case such as this, the facts of which speak for themselves. The chief feature of interest is the extreme rarity of pseudo-hypertrophic paralysis attacking an adult, this disease being as a rule almost entirely confined to children. In eighty cases cited by Eulenburg, only five commenced after adult life. Another point of great rarity is the extensive distribution of the disease, especially to the muscles of the neck, jaw, and tongue.

At first sight it may appear, in reference to the preceding measurements of the limbs, that they do not much exceed those of an ordinary well-developed man. But it must be remembered that the patient was short, evidently by nature of a slight and rather spare build, and whose original muscular conformation had been anything but highly developed. Inspection of the case made this very apparent, as there was so striking a contrast between the hypertrophied muscles and those unaffected, the former standing out prominently dense and hard, forming a deformity when compared with the non-affected muscles, and the small and slight osseous conformation. Taking the leg as an example, there was a small and almost delicate foot sparingly clothed with muscles, a thin ankle only measuring  $7\frac{1}{2}$  inches in circumference; and suddenly the mass of the gastrocnemius and soleus bulging out, being in girth  $15\frac{3}{4}$  inches, and then at the knee-joint the rapid decrease in size to a small and almost fleshless articulation. The deformity thus occasioned, together with the marked increase in size of the muscles, were more readily appreciated at the time than can now be described. The difference was also well marked in the face, which was, as a whole, thin and pinched-looking, with the mass of the temporals and masseters abnormally developed.

As has been already pointed out, exactly in proportion as the various muscles were increased in bulk, so was their vigour and power diminished, thus distinguishing this affection from true hypertrophy.

It is unfortunate that this patient's views of public morality were of so elastic a character, as not only had they previously subjected him to censure by the state, but compelled us summarily to eject him from the hospital after a few days' observation.

## CASE OF ACUTE TRAUMATIC TETANUS.

BY SURGEON-MAJOR J. J. L. RATTON, M.D., M.C.

*Madras Medical College.*

LUTCHMOO, aged 30 years, a married Hindoo woman, was admitted to the Hospital for Women and Children, Madras, on the 10th of May, 1879, with the following history.

*History.*—Two days before admission, that being the third and last day of a menstrual period, she took a cold bath. Next day symptoms of lock-jaw appeared, and the day after she sought admission to hospital.

*May 10th. State on Admission.*—The patient is a strong, well-developed woman. The muscles of her neck and abdomen are hard and contracted. Trismus is established, the mouth cannot be opened more than half an inch. She has occasional slight spasms of opisthotonos. She can swallow without much difficulty. The left ankle is disfigured with deep syphilitic ulcers, some with sinuses and tense cicatricial bands. Pulse 136. Temperature  $102^{\circ}2$  F. Respiration 30.

*Treatment.*—The cicatrices and sinuses were slit up to relieve tension, and a poultice smeared with belladonna ungt. was applied over the ankle. Calomel grs. v. and pulv. jalapæ grs. 30 at once. Chloral hydrate 3ss in syrup every two hours.

*May 11th.*—Patient's bowels have been moved, and she has taken 300 grs. of chloral. Trismus is further developed, and there is great difficulty in swallowing. The spasms of opisthotonos are decided, and occur about every hour. Patient is well under the influence of chloral, but not completely so. Evening temperature  $103^{\circ}$  F. Pulse 140. Respiration 32.

*Treatment.*—Continue chloral 3ss every two hours by the mouth as long as a patient can swallow, afterwards by the rectum. Strong beef-tea every three hours, by mouth or rectum.

*May 12th.*—Decidedly worse. Although completely under the influence of chloral, the tetanic spasms are excited by the least noise or motion. The body is arched forwards, the head

thrown back; every muscle of the trunk and extremities is firmly set; the teeth are clenched, and the patient remains thus, breathless, as if cast in iron, for sixty seconds or so. There is no mistaking the acute nature of the case. Evening temperature  $102^{\circ}8$  F. Pulse 136. Respiration 32. Nutrient and chloral enemata had been required for the past eighteen hours. Three hundred grains of chloral used.

Seeing that the patient was worse even with the very free exhibition of chloral adopted, I decided to give nerve-stretching a trial. The operation was performed at once.

*Operation.*—Under chloroform, and with the carbolic spray, an oblique incision was made in the left ham downwards, from the lower border of the gluteus maximus. There was some difficulty in finding the sciatic nerve, which was covered by  $2\frac{1}{2}$  inches of fat, and overlapped by muscle. When found it was drawn out of the wound and stretched with a force of about 30 lbs.<sup>1</sup> for the space of two minutes, and afterwards jerked once or twice with about double that force before being replaced. Silk sutures and boracic dressing.

*After the Operation.*—Not a single spasm occurred during the rest of this day for fourteen hours after the operation.

*May 13th.*—Patient well under the influence of chloral, but very restless, tossing from side to side. Slight spasms of opisthotonos appear to occur at intervals. Has had 300 grs. of chloral by the rectum since the operation. Temperature  $102^{\circ}5$  F. Pulse 144. Respiration 32.

*May 14th.*—The spasms have ceased altogether. Patient is quite under the influence of chloral, but can be roused to take nourishment, which she begins to swallow. Has taken 270 grs. chloral diet at this time. Beef-tea, 4 pts. (concentrated); milk, 4 pts.; eggs, 4; arrack, 6 ounces, by mouth and rectum.

*Treatment.*—Cont. chloral 3ss every three hours. Evening temperature  $102^{\circ}5$  F. Pulse 130. Respiration 36.

*May 15th.*—No spasms; under the influence of chloral yet. Very restless. Has taken 180 grs. chloral by the mouth. Sciatic wound begins to suppurate. Temperature, evening,  $102^{\circ}2$ . Pulse 136. Respiration 34.

*May 17th.*—Still chloralised. Not so restless. Muscular tension continues, but no spasms. Reduced chloral to 3ss every fourth hour. Temperature  $102^{\circ}$  F. Pulse 120. Respiration 30.

*May 19th.*—Improving. Trismus diminishing. Takes 120 grs. chloral daily. Not so restless. Temperature fell to normal this day, and continued so for the rest of the case. Pulse 120.

<sup>1</sup> The force required to lift a 3 lb. weight.

*May 22nd.*—Doing well. Passed a round worm by the mouth. Ordered santonine grs. v, followed by a purge. Bowels opened three times; no worms. Ordered chloral 3ss three times a day.

*May 29th.*—Can open her mouth about halfway. Can speak distinctly. Begins to masticate, and is ordered full diet at her own request. Neck and abdominal muscles still tense. Continue chloral 3ss twice daily. Sciatic wound healthy suppurating and dressed from the bottom.

*June 1st.*—Still improving. Chloral 3ss at bed time.

*June 23rd.*—Patient is able to sit up. Tetanus quite gone. Sciatic wound healing. Tried her again with santonine, but found no worms. Sensation, nutrition, and power equal in both limbs.

*Remarks.*—Notwithstanding the fact that a chill after bathing at the end of a menstrual period was the exciting cause of the attack, I think all will agree that the above case was one of traumatic tetanus. To those who would call it puerperal, I may suggest that traumatism is concerned in the origin of puerperal tetanus. This woman had never borne children. She was strong, stout, and in the prime of life. Although well saturated with chloral, the symptoms rapidly developed into an explosion of acute tetanic violence, which would surely have carried the patient off but for the nerve-stretching. At the same time, nerve-stretching would not have saved the patient but for the chloral. Eighteen hours after the operation slight spasms reappeared, and if at this time chloral had not been pressed, I fear the spasms would have gathered strength. The day after the operation I left instructions at the hospital to send for me if clearly marked spasms of opisthotonos returned, as I intended to have opened the wound and stretched the nerve again. This is the fifth time that I have stretched the sciatic for traumatic tetanus, and each time the separation has suppressed spasm for many hours. If during these valuable hours of rest chloral is properly plied, the chances are that spasm will not return. I have been obliged to give chloral very freely, and to maintain narcotism for about ten days. During the three critical days, on and after the operation day, the patient had 300 grs. chloral daily. The absolute fatality of acute tetanus justifies the exhibition of the drug in what appears to be a reckless fashion. There is besides an astonishing toleration of chloral. The sciatic wound suppurated freely, but there was no pain complained of in the limb, and there was no contraction of it. In this case I purposely refrained from operating until the symptoms were alarming, for I wished to test the value of nerve-stretching in



acute cases only. Sub-acute cases get well of themselves. Really acute cases, I believe, never get well of themselves. It would be well whilst this treatment is on its trial to wait for an outburst of acute symptoms before operating. We shall then sooner know the value of the remedy than if it is used indiscriminately, or before it is quite certain that the case is going to be acute. I doubt if any treatment will avail to cure acute tetanus, unless age and stamina are with the physician. I mean by age, about thirty years, the prime of life. In this case the duration of the disease was twenty-three days. It is seldom less in acute cases. Only the strong can hope to battle through the exhausting efforts of clonic and tonic tetanic spasms prolonged for days, generally with insufficient nourishment, often, for days at a time, with rectal alimentation only. Yet, considering the well-merited reputation of tetanus as a fatal disease, it seems to me a solid gain that even a few cases may in certain circumstances be saved by modern medicine.

## Abstracts of British and Foreign Journals.

**Report on Visceral Neurology.** "TROPIC NERVES."—In this Report for January (see Vol. I. p. 581) an account was given at some length of the experiments of Steiner upon the effects of section of the cervical vagi on the nutrition of the lungs. Steiner confirmed Traube's view, that the pneumonia that follows this operation is traumatic and not trophic in its nature, and is proximately due to paralysis of the pharynx, larynx, and associated parts. Michaelson has studied the same subject (*Centblt.* 1879, No. 22, p. 390), separating as far as possible the influence of the superior laryngeal from that of the vagus proper. He concludes that the changes in the lung consequent on section of the vagus proper are immediate; that they affect chiefly the lower and middle lobes; and that they are of the nature of œdema, hyperæmia, and splenization, with hæmorrhage and emphysema. By direct inspection Michaelson observed within a few minutes after section, an elevation of temperature in the lungs to the amount of  $\cdot 25$  C.; and redness of the pulmonary tissue, which reached its height in half an hour, and then became complicated with hæmorrhage. On the other hand, the changes that follow section of the two recurrent nerves are chiefly located in the upper parts of the lungs; consist of a catarrhal pneumonia, which generally becomes caseous; and do not make their appearance until the laryngeal paralysis has exerted its injurious influence for a sufficient length of time. Michaelson concludes that the vagus contains vaso-motor fibres for the lungs.

It may be remembered that in the paper referred to above, Steiner attributed the death of the animals experimented on in this way to the inflammation of the lungs. A remarkable amount of attention appears to have been recently devoted in Germany to the subject of vagotomy, and an active controversy has been maintained between several observers on the point just mentioned.

Eichhorst, in a special work (reviewed in the *Centblt.* 1879, No. 10, p. 181), refers the death of the animals (birds) after section of both vagi to cardiac paralysis from fatty degeneration of the myocardium, which he considers a purely trophic lesion. This author therefore concludes that the vagus contains trophic fibres to the heart. Thus the question of the trophic function of the vagus, which seemed to be settled in the negative as regards the lungs, is now reopened on fresh ground, namely, as regards the heart. Zander of Königsberg opposes Eichhorst's view, and states (*Centblt.* 1879, No. 6, p. 99; and No. 7, p. 115) as the result of more than eighty experiments on birds, that the cause of death after vagotomy is starvation, with which fatty degeneration of the heart and cardiac failure are associated as *effects*. The latter point is confirmed by Wassiljew (*Centblt.* 1879, No. 21, p. 382); who, however, in a subsequent contribution (*Ib.*, No. 27, p. 494) states that injury to the cervical vagi in a rabbit produced after the lapse of several months fatty degeneration of the heart. Zander's position is however much more seriously assailed by Eichhorst, who contends in reply (*Centblt.* 1879, No. 10, p. 161) that in animals dying of starvation the change in the muscular tissue of the heart is not true fatty degeneration (such as is found after vagotomy) but a granular dulness—the *trübe Schwellung* of Virchow, which Zander had probably mistaken for the former lesion. Eichhorst maintains, therefore, that, after double vagotomy, birds die not of inanition, but of acute fatty degeneration of the heart, of trophic origin.

A subject which is closely related to the preceding is the localisation of acute disease of the respiratory organs in hemiplegia, to which Rosenbach has drawn attention. (*Centblt.* 1879, No. 16, p. 282.) This author believes as the result of his observations that the paralysed side is much more frequently than the sound side the seat of acute lung-disease. In searching for an explanation of this striking observation, Rosenbach found that both the direct and the reflex irritability of the involuntary muscular tissue of the paralysed side is decidedly lowered. One result of this condition will be that foreign matter (saliva), entering the bronchi of the corresponding side, will not give rise to sufficient expulsive efforts; and that unilateral broncho-pneumonia will be set up. The author withholds any explanation of the occurrence of croupous pneumonia and pleurisy under the same circumstances.

Professor Hagen has contributed to the subject of trophic lesions, the results of experiments upon the effect of section of the trunk,

of the fifth nerve within the skull, on the condition of the mucous membrane of the tympanum (*Archiv f. Experim. Path. und Pharm.* xi., 1 & 2, p. 39). These results are, however, as various and conflicting as the conclusions that have been previously drawn from similar experiments connected with the cornea; so that the question of the effect of lesion of the trigeminus on the nutrition of the parts that it supplies appears to be as far from being settled as before. Gellé had concluded (*Gaz. Méd. de Paris*, 1878, No. 1) from a single experiment on a dog, that the trigeminus contains trophic nerves to the mucosa of the middle ear, inasmuch as a lesion of the medulla oblongata involving the root of the fifth nerve was followed not only by inflammation of the eye and nose, but by suppuration of the corresponding tympanum, the animal having lived about ten days. Professor Hagen repeated this experiment upon thirteen animals, but instead of injuring the medulla, cut the trigeminus on one or both sides without opening the skull. In eleven of the cases not a trace of exudation was present in the tympanum. In the remaining two cases exudation was found after twelve hours and thirty-one hours respectively. In one of them there was also abundant sero-sanguinolent non-purulent fluid within the *opposite* tympanum—an appearance which Hagen (perhaps somewhat hastily) considers a proof that the inflammation was not on either side to be regarded as a trophic lesion. The presence of exudation in the second case he explains as accidental, and due to other causes (!). In one of the “negative” cases, although no “exudation” was present, there was found when the animal was killed, some sixty days after section of the right trigeminus, a perforation of the membrana tympani, which was stopped by a whitish-yellow tallowy mass. When these results are considered, it is somewhat surprising to find that Hagen concludes that the integrity of the tympanum after section of the trigeminus conduces to “completely support the traumatic nature of ‘trigeminus-keratitis.’”

A case of joint-change in consequence of lesion of the nerves of the hand is reported by Bouchut, who considers it “trophic” in origin. A child which had the distal phalanx of the right thumb crushed at the point six years previously, presented, at the age of 7, atrophy of the muscles and bones of the right forearm and hand; pains in the joints of the hand and fingers; and knobby prominences on the distal joints of *both* hands (*Centblt.* 1879, No. 12, p. 224).

THE HEART.—The attempts to discover a “centre” for the heart in the cerebral convolutions, that is, a point or region which could

be proved by faradisation to influence the cardiac rhythm, have not been successful in the hands of Eckhard (*Centblt. f. d. m. W.*, 1878, No. 34, p. 611). In non-curarised animals, faradisation of the cortex cerebri produces, as a rule, no effect upon the beat of the heart, so long as there are no sudden movements of the body. If sudden movements occur, whether from direct irritation of the so-called "motor" centres or from other excitation, then the heart and circulation may be affected. The rule is that no cardiac disturbance can be discovered as long as only single movements of the opposite anterior extremity are induced. If other movements occur, the pulse becomes less frequent and stronger (with weaker beats between), and that through the medium of the vagus. At the same time the blood-pressure rises somewhat, and that independently of the heart. Irritation of the *cornu ammonis* was found to have no influence on the cardiac movements, such as Balogh has described, so long as no muscular movements were induced.

Ludwig (of Pontresina) and Luchsinger publish (*Centblt.* 1879, No. 23, p. 404) the results of their researches on several points connected with the innervation of the heart, and especially with reference to the activity of the intracardiac ganglia. They find that the ganglia are paralysed by high temperatures, while their irritability is again restored on cooling. Just before the onset of paralysis, and again as soon as the impulse is restored, the vagus is irritable to a high degree. The inhibitory elements of the heart are therefore more resistant to heat than the motor elements are. The activity of the vagus falls as the intracardiac pressure rises, for the excitation of the motor elements increases with the pressure. Therewith also rises the frequency of the pulse; and this both as regards the heart as a whole, and portions of the heart artificially separated.

THE VAGUS.—The view of Arloing, Tripier, and Masoin, that the heart is more powerfully affected through the right vagus than it is through the left, that is, that the right vagus essentially contains cardiac fibres (whilst the left vagus essentially contains respiratory fibres), has not been confirmed by Langendorff (*Centblt.* 1879, No. 23, p. 406). The truth appears to be that the results obtained from galvanisation of both nerves are extremely variable.

A case of exceedingly frequent pulse (136–148 per minute, persistently) is recorded by Meixner (*Centblt.* 1879, No. 31, p. 573), in which the left vagus was found *post mortem* to be compressed by an enlarged and cheesy lymphatic gland.

The subject of vagotomy has been discussed above under "*Trophic nerves.*"

VASO-MOTOR CENTRES AND NERVES.—The view that is held by some physiologists that dilatation of the blood-vessels may be brought about by vaso-dilator nerves connected with vaso-dilator centres in the spinal cord, is supported by certain experiments of Dr. Ott of Baltimore (*Journ. of Physiol.* II. i. p. 51). He has determined that in the cat a centre of the vaso-dilator nerves for the skin of the posterior extremities is located between the tenth dorsal and the first lumbar vertebrae.

Jolyet (*Gaz. Méd.* 1878, No. 46) has found by experiments on curarised dogs that faradisation of the trunk of the superior maxillary branch of the fifth nerve of one side in the pharyngo-maxillary fossa, causes active hyperæmia, with elevation of temperature of the mucosa of the nasal fossæ and of the gums; of the mucosa and skin of the upper and lower lip; and of the skin of the ala of the nose of the same side. These effects are not produced if the vago-sympathetic trunk of the same side have been previously cut. Further, section of the superior maxillary is followed by slight hyperæmia of the above-mentioned parts, which is reduced by irritation of the peripheral end of the divided nerve. According to Jolyet, these results indicate that the vasor dilatation in the parts is due to reflex paralysis, through the trigeminus, of vaso-motor nerves contained in the sympathetic. This view is supported by the fact that faradisation of the superior maxillary of one side (with, as well as without, section of the corresponding sympathetic in the neck) also produces similar hyperæmia of the skin and mucosa of the opposite side, though to a less degree.

Vaso-motor fibres to the lungs are referred to under the head of "*Trophic nerves.*"

RESPIRATION.—The histological characters of the nerves of the lung in the frog and calf have been investigated by Egorow, working under Chrzonszczewsky. In the frog, the great bulk of the nerve fibres entering the lungs are non-medullated. The trunks of these are provided with abundant ganglia, the cells of which mostly present the well-known "spiral fibres" of Beale. The medullated fibres enter the muscles of the alveolar septa; lose their medullary sheath; and, forming a network, end in the muscular fibres. The non-medullated fibres breaking up into fine and finer networks; end in the sub-epithelial muscular layers of the alveoli; and extend into the pleura. The vessels are supplied by the non-medullated fibres (*Centblt.* 1879, No. 18, p. 305).

Langendorff appears to have succeeded beyond other observers in obtaining experimental evidence of the existence of "expiratory," or "inhibitory" or "slowing" fibres to the respiratory centre, in the trunk of the vagus (*Centblt.* 1879, No, 21, p. 375). While Rosenthal and others found that a strong current applied to the central ends of the divided vagi causes acceleration of respiration, with finally tetanus of the diaphragm (extreme inspiration), and only occasionally slowing of respiration and arrest in expiration, Langendorff observed expiratory almost as often as inspiratory standstill. Mechanical and thermic stimuli in the same way caused respiratory slowing; which was even more pronounced by chemical irritation. No support was found for the view that inspiratory and expiratory fibres run chiefly in the one and in the other vagus respectively; nor for the view that the left vagus contains chiefly respiratory fibres (and the right vagus chiefly cardiac fibres). Langendorff also concludes that the respiratory centre is double, from the fact that acceleration of respiration may be induced by stimulation of one vagus after exhaustion of the other vagus by protracted stimulation. Finally, this observer believes from his experiments that the slowing and accelerating influence on the respiration of irritation of the vagus is not peculiar to this nerve; but that many, and probably all sensory nerves possess this property, slight excitation accelerating the breathing, and powerful excitation slowing it.

PERSPIRATORY CENTRES.—Dr. Ott in his investigations upon the physiology of the spinal cord (*Journ. of Physiol.* II. i., p. 51) endeavoured to localise the "perspiratory centre" of the hind foot (in the kitten); and confirms the view of Luchsinger that it is situated in the lower dorsal part of the spinal cord, whence the nerves pass to the limbs through the abdominal sympathetic and sciatic trunks. The sweat-nerves of the anterior extremity appear to emerge from the cord in the fourth dorsal nerve, and to pass through the stellate (or first thoracic) ganglion to the brachial plexus. Thus in both extremities the secretory nerves appear to follow closely the course of the vaso-motor fibres. Sweat-centres, like vaso-motor centres, are also situated throughout the cord and medulla oblongata; for irritation of the latter causes abundant perspiration in all the extremities. An inhibitory sweat mechanism also appears to exist, and to be partly peripheral (multipolar nerve-cells in the glands,) and partly central—the fibres to the lower limbs running in the sciatic and abdominal sympathetic from the cord, and perhaps from the medulla.

Dr. Ott also found that the stimulant effect of asphyxia, i.e. of excess of carbonic acid in the blood, upon the sweat-glands, is exerted through the centres in the cord. The same applies to the stimulant effect of heat upon the perspiratory glands, which is not direct but central. On the other hand, pilocarpin appears to excite perspiration through a peripheral mechanism; and muscarin by direct stimulation of the secreting cells.

With respect to the *path* of the perspiratory fibres in the spinal cord between the lower extremity of the dorsal region and the medulla oblongata in the cat, Ott confirms the results of previous observers in the rabbit; namely, that the secretory fibres connecting the sweat-centres with each other run in the lateral columns, at least in the dorsal portion of the cord, about the sixth and seventh vertebræ. The sweat-fibres appear not to decussate in the case of the cat.

INNERVATION OF THE PELVIC VISCERA.—Dr. Ott (*loc. cit.*) determined several points of interest with respect to the rhythmical contraction of the sphincter ani under nervous excitation, and discovered a similar rhythmical contraction of the sphincter vaginae. The ano-spinal and vagino-spinal centres were determined to lie together between the sixth and seventh lumbar vertebræ. He made the further observation that irritation of the vaginal or rectal mucosa may induce rhythmical adduction and extension of the voluntary muscles of the posterior extremity of one side. The *vesico-spinal* centre is placed by Dr. Ott (as by Kupressow) between the fifth and sixth lumbar vertebræ (cat and rabbit). The *thalami optici* were proved to contain in part at least the centres of the inhibitory apparatus presiding over the ano-spinal and vagino-spinal centres, and restraining their rhythmical action.

LIVER AND PANCREAS.—In Zander's experiments upon the effects of section of the vagi, described above, the observation was made that the liver continued to secrete abundance of bile, and that the pancreas was most probably also active. Glycogen and sugar disappeared very soon after the operation. It must not be forgotten, in estimating the significance of these results, that the animals (birds) were dying of inanition from complete paralysis of the oesophagus.

J. MITCHELL BRUCE.

**Locomotor Ataxy, and its Connection with Injuries.**  
(*Revue Mensuelle de Médecine et de Chirurgie*, No. 3, 1879.)—Dr. L. II. Petit endeavours in this paper to throw light on the in-



fluence which traumatic lesions have on the origin or course of locomotor ataxy. It will readily be conceded that he gave himself a somewhat difficult task, for the records of cases have been so often meagre and uncertain as to make them more or less unsuited as reliable data for inquiry. Few questions, indeed, are less easy to decide than the influence of injuries in giving rise to slowly progressive diseases of the nervous system. The insidious appearance of the symptoms, and the often long duration of those which are less obvious and which may have been at first transitory and occasional, render it impossible perhaps to say when the particular disease had a beginning. An accident, or an acute illness, may have been the one thing needed to upset a balance which had hitherto been maintained, and to reveal conditions hitherto unsuspected or unobserved. Examples of this are met with every day in diseases where the nervous system plays but little part, and if mistakes are made in simpler cases, how much more readily may error arise as to the causation of those diseases of the central nervous system itself, whose earliest manifestations are hidden and obscure! And more especially may this be said of locomotor ataxy.

For the purposes of his inquiry M. Petit gathers together a large number of cases from French, German, English, and other writers, and his long and cautious essay is a valuable contribution to the study of an important subject. Viewed, however, with no undue scepticism, we cannot but think that grave doubts must rest upon much of the evidence which many of the cases appear to show, and that the *post* and the *propter* have often been confounded.

To take the first case recorded in 1844 (W. Horn, in Steinthal, *Journal de Hufeland*) we read as follows: "Man, aged 37. Fall from horse; unconscious several hours. No morbid symptoms afterwards; but, in the winter following, onset of pains and cramps in the lower limbs; after that numbness in the legs, gait uncertain and tottering; dysuria with incontinence. Horn diagnosed a *tabes dorsalis* of traumatic origin. Bleeding and strychnia without result." A history like this, and it is neither more nor less precise than many histories in the paper, is inadequate, we think, for the foundation of any very decided opinion, notwithstanding the fact that Steinthal, who gives the case in his paper, "*Beiträge zur Geschichte und Pathologie der Tabes dorsalis*," attached great importance to the traumatic origin of the *tabes* in this particular instance. True, the author himself seems to entertain much the

same objections to his collected matter, for he writes that the evidence therefrom is hardly sufficient to warrant any precise conclusions, and he would rather point out the channels in which further inquiry may fitly flow. Better than by searching medical literature for imperfect records in the past, let greater care be bestowed on the observation and record of future cases, remembering that here, at least, conjectural statements must not fill up gaps in evidence, if there is to be a right estimate of the weighty matters which pertain to the history of disease.

In addition to the influence of injuries in the production of locomotor ataxy, M. Petit examines that of traumatic lesions, accidental or other, upon the course of the already existing disease; the influence upon it also of intercurrent febrile affections, e. g. pleurisy and erysipelas; and finally, the influence of locomotor ataxy itself upon the progress of traumatic lesions.

Here it must suffice to give the conclusions at which the author has provisionally arrived. Direct or indirect injuries of the spinal column, falls on the back, the buttocks, or the feet, give rise to shake of the spinal cord, and in consequence thereof to lesions which may become the starting-point of chronic myelitis, and induce the symptoms of locomotor ataxy. It is impossible so far to affirm that injuries of other and distant parts have the same pathogenic influence, although it is probable that in persons predisposed to general sclerosis, the arthritic, the syphilitic, the alcoholic, such injuries may, by irritation of the spinal marrow, hasten the development of ataxy. Injuries of this nature can certainly revive an ataxy apparently cured, or hurry on the course of one already existing; while various morbid actions, the suppression of habitual secretions or discharges, of hæmorrhoidal or catamenial flow, or of cutaneous eruptions, pregnancy, and intercurrent febrile affections, seem to act in a like manner.

Ataxic symptoms may be localised in some regions or organs affected either before or after the appearance of ataxy. In some instances prolonged peripheral irritation has given rise to nervous phenomena which have been wrongly described as locomotor ataxy, and which have disappeared with removal of the cause. Associated as locomotor ataxy often is with derangements in the nutrition of certain tissues, it can modify the course and progress of local lesions, an observation pointed by the consideration of cases of centused joints, fractures, and laceration of soft parts.

**Cases of Bulbar Paralysis.** Prof. W. ERB (*Archiv für Psychiatrie*, vol. ix. Pt. II.).—The interest and importance of the electrical reactions of nerve and muscle in various diseases are becoming daily more evident, though the ridiculously inadequate methods of investigation prevalent among us still prevent electrodiagnosis from occupying the position it deserves. The first case presented by the author forms the theme of some important remarks upon the electrical irritability in bulbar paralysis. He had already propounded his views in Ziemssen's *Cyclopaedia* (vol. xi.); but here he adduces a body of confirmatory evidence. Clinical facts, as well as experimental, have shown that *qualitative* changes in electro-muscular reaction occur only in such lesions where we must assume an interference in the "trophic" influence of the cord upon the nerves and muscles. Such changes are observed in their full perfection in cases of "severe" facial paralysis, or of traumatic paralysis of various nerves. But in these cases the nerve itself is always found to have undergone change, and lost its electrical irritability. Now in bulbar paralysis, and in a whole series of cases such as poliomyelitis, progressive muscular atrophy, &c., it is sometimes found that the muscles present the characteristic reactions of degenerative changes, whilst the nerves (at least during the earlier period of the disease) react normally or almost normally to both currents.<sup>1</sup>

Prof. Erb draws attention in the same case to the phenomenon of increased reflex in the facial muscles, which responded readily to slight stimulation of the superjacent skin, the nose and the eye.

The second part of the paper describes a case which presented, in addition to well-marked bulbar symptoms, phenomena of paresis and pain in the extremities, double vision, intense headache, noises

<sup>1</sup> In the 'British Medical Journal' (May 31st, p. 837) an editorial note appended to a letter of mine objects that the cases are yet too few to prove that this modified form of degenerative reaction necessarily points to a *central* disease. But, first, what is claimed for it is simply that it points to a "spinal" paralysis in Marshall Hall's sense. Secondly, the objector forgets that in this modified form there occur qualitative as well as quantitative disturbances in the muscular reactions: a phenomenon absolutely unknown in purely local muscular disease.

Recently I had an opportunity of diagnosing a case of progressive muscular atrophy, and one of polio-myelitis anterior chronica in their incipient stages, in which only local disease was supposed to exist. The further progress of the cases justified the diagnosis. I have now under observation a case of bulbar paralysis where the lip muscles present the reaction of A C C > K C C (both diminished) with almost normal nervous irritability.

in the ears, weakness of the masticatory and cervical muscles, &c., and which against all expectation made a fair recovery after a course of galvanic applications to the head and neck. The marked influence of the very first sittings leaves no room to doubt the efficacy of the treatment.

The third part treats of a new, and probably bulbar, "symptomen complex." Under this heading three cases are described in which the leading symptoms were (1) Ptosis; (2) Paresis of the muscles of the back of the neck; (3) Paresis of the masticatory muscles.

The development of these symptoms took place in a few months, accompanied with pain in the neck and head, and with vertigo in two cases. There was also weakness of the extremities and of the tongue, but no change in the lower half of the face; whilst in two cases the upper half was slightly paretic with increased irritability. Other symptoms occurred in the three cases severally, interesting but not sufficient to veil their fundamental unity.

One of the patients died rather suddenly. Unfortunately she had left the hospital, and no autopsy was made.

**Conclusions from the Study of 125 Cases of Writers' Cramp and Allied Affections.** Dr. G. M. BEARD (*New York Medical Record*, March 15, 1879).—In this paper the author gives a summary of what will form the subject of an ulterior publication.

1. The name of the disease is a misnomer; often cramp occurs only late (sometimes never) in the progress of the complaint. Among the numerous other symptoms which characterise it are: fatigue, aching pain, nervousness, trembling, stiffness, paresis or paralysis, numbness and numerous other subjective sensations, abnormal grasp of the penholder, &c. These symptoms often extend beyond the hand and arm.

2. In other professional "cramps," such as that of musicians, telegraphists, painters, dancers, blacksmiths, barbers, &c., no one symptom is diagnostic.

3. The author's view of the pathology is that the disease is primarily of peripheral origin; secondarily and rarely it becomes central.

4. Writers' cramp occurs chiefly in those whose constitution is vigorous. In the nervous it is less common, and then is more easily relieved.

5. It occurs rather in consequence of routine work (copyists, &c.) than of work implying thought (authors, &c.).

6. It is no longer an incurable disease. The chief agents in its treatment are galvanism and subcutaneous injections of atropia, strychnia, duboisia, arsenic (pushed to their physiological effects). To these may be added massage, blisters, cautery; and internally calabar bean, ergotine, iodoform with nerve foods.

The author describes a number of devices by which the symptoms may be palliated, chiefly modifications in the making or holding the pen.—Some observations on the speed of handwriting are appended, and the paper concludes with some remarks on telegraphers' and musicians' cramp.

A. DE WATTEVILLE.

**Dimensions of the Fœtal Head.** (*Gazette des Hôpitaux*, No. 89, July 15, 1879.)—At the Société de Biologie, MM. Budin and Ribement have given the results of their observations of 211 cases. The heads of that number of infants at the age of 40 to 72 hours have been minutely examined, for then the cranium, deformed by the pressure of parturition, had returned as nearly as possible to the fœtal condition.

These measurements were soon found to vary with the weight of the infant; it was therefore necessary to study these points in connection. And having had to distinguish two series (male and female), each series has been divided into six classes.

		Gr.	Gr.
Class A.	Infants weighing from	1500	to 2000
„ B.	„ „ „ „	2000	„ 2500
„ C.	„ „ „ „	2500	„ 3000
„ D.	„ „ „ „	3000	„ 3500
„ E.	„ „ „ „	3500	„ 4000
„ F.	„ „ „ „	4000	„ 4500

Class D. (gr. 3000 to 3500) may be considered as “normal,” and the results of measurements of males and females coming within it are subjoined.

1. Maximum diameter, chin to farthest point of cranium on the sagittal suture,  $13\frac{1}{2}$  c.

2. Occipito-mental diameter, 13 c.

3. Occipito-frontal diameter (root of nose),  $11\frac{3}{4}$  c.

4. Diameter from the point of meeting of the occipital with the neck to the middle of the great fontanelle, at the level of the point where the sagittal and parieto-frontal sutures cross, 10 c.

5. Biparietal diameter, or greatest width posteriorly,  $9\frac{1}{2}$  c.

6. Bitemporal diameter, or least width anteriorly; from the rise of the parieto-frontal suture on one side to that of the other,  $8\frac{1}{4}$  c.

7. Bimastoid diameter,  $7\frac{3}{4}$  c.

Greatest circumference at the plane of the greatest antero-posterior diameter, 38 c. 12. A smaller circumference on the plane of number 4, 32 c.

In comparing the figures obtained in the different series, MM. Budin and Ribement have found that the diameters are increased in proportion to the weight of the foetus, that it is not the sex but the weight that causes variation in the size of the head.

The length of the body increases with the total weight of the infant. They also found with regard to the male infants the figures 41 c. 3, 45 c. 7, 47 c. 1, 49 c., 50 c. 2, 51 c. 1, corresponding to the classes A, B, C, D, E, and F. Among the females there was an analogous progressive increase.

To conclude, the increase of the diameters of the head and the increase of the length of the body are proportionate to the weight of the foetus. Taking the Class B, weighing a little more than gr. 2000, and the Class F, weighing a little more than gr. 4000—that is nearly double—they find the diameters of the head of the last series measure one centimetre, sometimes two centimetres more.

A. R. URQUHART.

**American Neurological Notes.** J. H. SCARFF, M.D., publishes (*Virginia Medical Monthly*) four cases showing the value of chloral hydrate in obstinate vomiting of pregnancy. The method of administration is, 20 grains in solution night and morning, till 4 doses have been administered. In the same Journal chloral is referred to as having been successfully employed in cases of retention of urine, also associated with pregnancy. What we already know of chloral leads us to anticipate that in all such reflex-neuroses the drug is likely to be of great service.

Dr. Cullen, quoting from the *Italian Medical Gazette*, Venice, states that in hysterical and hypochondriacal affections camphor has been shown to be a powerful hypnotic. This statement, which many who have used camphor largely without arriving at such a favourable conclusion may be inclined to doubt, may have been justifiably made, as the camphor was dissolved in olive oil and administered hypodermically. It is highly probable that this time-honoured

drug has recently suffered much in reputation, owing to the difficulty experienced in exhibiting it in a soluble form. Camphor dissolved in olive oil and administered hypodermically might be well worthy of a careful trial in the sleeplessness of simple mania.

Kunze treats cases of epilepsy of long standing with curare. He uses a solution of 7 grains of curare in 75 minims of water, to which he adds two drops of hydrochloric acid. At intervals of about a week he injects subcutaneously about 8 drops of this solution. In various cases in which convulsions had occurred for several years, he obtained "a complete cure" after 8 or 10 injections.

Dr. T. Kennald publishes in the *New Orleans Medical and Surgical Journal* some excellent observations on sunstroke, based mainly on the 1873 epidemic in St. Louis. The fatal temperature began somewhat suddenly on July 10th, and continued without much intermission for about ten days. During this period the total mortality from all causes was 499, and 154, or 31 per cent. of these deaths was caused by sunstroke. It is notable that many more deaths were assignable to extreme heat, though not directly due to sunstroke. Thus there were 50 from *cholera infantum*, 17 from apoplexy, 40 from convulsions, and 34 from other diseases of the brain and nervous system.

Nervous exhaustion from exposure to an excessive degree of heat is maintained to be the true meaning of sunstroke, and humidity of the atmosphere is not admitted to have any share in the production or prevalence of the stroke. Neither is an intensely dry atmosphere required, the sole requisite being the uninterrupted continuance of high temperature.

Dr. Kennald does not seem to have directed his attention to the question of modifications in the potency of chemical rays as distinguished from mere high temperature; but he states that after the commencement of an epidemic many deaths occur on cloudy days in the shade, where the temperature does not range very high. "Persons debilitated by age and worn out from grievous sickness are often overcome by heat when they have not been exposed to the sun's rays at all, which proves that death is due to nervous exhaustion from the heat, and not to insolation." To some who have suffered severely in spirits and prospects from the prolonged absence of sunshine, the study of mortality from sunstroke may yield a lugubrious form of satisfaction.

Dr. Daniel Clark comments (*Canada Lancet*) on a statement said to have been made by Dr. Hammond, that he was in favour of punishing insane people just as he would a tiger who went about destroying people. If a lunatic had a homicidal mania, he would hang him. He would hang lunatics who had a mania to kill, even were that deed not performed. Dr. Hammond, if he is rightly reported, treads on dangerous ground. If such are his views, would he not be regarded by many as having a "mania to kill"; and run the risk of being treated like an American Haman?

ROBERT LAWSON, M.B.



# B R A I N.

JANUARY, 1880.

Original Articles.

## CHRONIC MORPHINISM.

BY H. OBERSTEINER, M.D.

*Privat-Docent in the University of Vienna.*

WHILE the progress of science is ever discovering new methods of combating disease, the advance of civilisation brings with it new enemies. Railways bring in their train spinal concussion, telegraphs develop telegraphic-cramp, and many valuable remedies become by their abuse the sources of new ailments. Such are the various forms of chronic poisoning, the prototype of which is chronic alcoholism, faithfully portrayed by Seneca. Specially worthy of notice in this relation is the continued abuse of chloroform, and opium and its alkaloids, more particularly morphia. It is different with nicotin, which is taken without the slightest evil effect by a large percentage of mankind; or even with arsenic. Chloroform and morphia differ from alcohol, inasmuch as the latter may be taken in considerable quantity without appreciable injury, so far as the nervous system is concerned; whereas the former two very frequently produce evil effects, and lead to earnest desire on the part of the individual to shake off the habit, which is at all times difficult and often impossible.

I will here confine my remarks to chronic morphinism, which is more common than chronic chloroform-poisoning. It

has to us a particular interest, inasmuch as quite an incredible number of our colleagues have fallen victims to it; and many have only just escaped. If medical men are charged—and it is to be feared, justly—with the propagation of this disease, owing to their carelessly, or for mere convenience' sake, leaving morphia and a subcutaneous syringe with the patient, it may be regarded as their punishment that the demon morphinism finds among them his favourite victims.

The number of cases hitherto published is not so great as to render unnecessary the description of others, yet they are sufficiently numerous to afford a tolerably accurate picture of the disease, and to indicate its treatment and prognosis.

As it is not my intention to write an exhaustive monograph on morphinism, I will content myself with calling attention to a few points, on the basis of my own and other observations, and in particular to those which at present are less generally known. The first commencement of the use of morphia is almost always some painful ailment; and then, as the pain continues, or even though it may have disappeared, the habit is formed, and more and more of the drug is demanded.

So began one of my patients, after traumatic injury of the sciatic; another in consequence of periostitis; a third (a physician) for toothache; a lady took to subcutaneous injections to allay violent pains in the sacrum caused by pelvic disease. In other cases, however, purely nervous symptoms lead to the formation of the habit. As is well known, we have in the subcutaneous injection of morphia a powerful weapon against feelings of fearful anxiety; so that states of melancholia, even of high degree, not unfrequently give way before it. Such intense anxiety and insomnia led in two of my patients to the use of morphia. But other nervous conditions lead to a similar result. Thus a lady exhausted by long-continued vomiting of nervous origin, found opium the only remedy of any avail. Though the existence of some painful affection is the chief cause of morphinism, yet there are others. A young medical man gave the following account of his own case. While he was attending the hospital, a patient was dismissed suffering from carcinoma of the stomach, and who had been for a long time treated with subcutaneous

injections of morphia. Next day the patient returned in a state of great excitement, and piteously begged for an injection, as otherwise he must die. This occurred in 1869, at a time when chronic morphinism and its phenomena were less known than now. As the physician was inclined to believe that the patient was romancing, he tried the experiment on himself to ascertain what the effects were. The result was that he formed the habit of morphinism, and never could overcome it.

With reference to the *quantity* taken, it is to be noted that it varies within certain limits, and may not exceed the maximum. The smallest quantities which can be taken continuously without any very marked effects, or reaction, are variable with the individual. I have under treatment a lady who has taken daily for more than a year about 0·003 gramme, and who has never felt inclined to increase the dose, though she strongly objects to giving it up entirely, on account of restlessness, sleeplessness, &c. While in most cases of continued morphinism a habituation occurs, so that more and more may be taken, there is a limit, variable in different individuals, beyond which, as a rule, it is impossible to go. If the morphinist has reached the maximum dose, he is aware of it, and instinctively keeps on this side of it; so that we very seldom hear of acute poisoning with morphia in such cases. There are individuals who never exceed 0·005 gramme daily, and have similar symptoms to those who consume 2–2·5 grammes. More than 3·5 grammes may not be taken daily continuously. This appears to be the limit, even in the most pronounced cases.

With respect to the symptoms of morphinism, they are so well known that they do not require to be detailed at length. If a morphinist who comes for advice and help is asked what symptoms he chiefly complains of, the answer is much the same in all cases. Disinclination to exertion in general; apathy; loss of memory; restlessness; generally also loss of appetite, various pains, hyperæsthesia; and in pronounced cases, suicidal tendencies. The patients are feeble, emaciated, have an ashy-grey or livid complexion, yawn and sneeze a great deal, and are impaired as to sexual power or completely

impotent. Less attention has been paid to the psychical condition. In this there is a certain analogy between alcoholism and morphinism. As the former is characterised by a moral deterioration, or moral starvation—whence the name formerly given, “*inhumanitas ebriosa*”—so in the morphinist there is a moral decadence in many cases. As he endeavours to conceal his habit in every way, so he loses his respect for truth and tells lies to conceal his condition, and afterwards from mere habit. He even lies to his physician; one day he says he has been accustomed to take 0·5 gramme daily, and next day he makes it 1 gramme. From this tendency to lying, which, however, is not all to be put to the account of morphia, he soon loses all self-respect; he feels how deep he has sunk, and, seeing no means of escape, he sinks deeper and deeper. This is, of course, not true of all cases; but knowledge of this fact is necessary in order rightly to estimate the utterances of morphinists.

The phenomena of the *morphia-hunger* vary with the individual and with the duration of the fast. Very often, where the quantity is slowly diminished, there are, with the exception of increasing discomfort, no violent symptoms. When, however, it is entirely withdrawn, or nearly so, violent symptoms occur. These are acute diarrhœa; insomnia; great excitement, or even mania, dangerous to those around, and particularly the physician; pains, itching of the skin, perspirations, a feeling of coldness, hallucinations, collapse, &c. In general, the gradual withdrawal has little advantage over the sudden. When great care has to be exercised on account of complications (great weakness, great excitability, &c.), it may be had recourse to rather than the other; yet, as has already been remarked, the explosive symptoms may occur nevertheless.

To relieve the sufferings of the worst period in the weaning process, various methods have been recommended; but unfortunately, though often useful, they are not so in all cases, e. g. wine in tolerably large amount, bromide of potassium, warm baths. In some cases, packing in lukewarm wet sheets was of great service. It may be remarked also that, when the drug is gradually withdrawn, it is often of advantage to inject the

subcutaneous same quantity of liquid as before. Occasionally even the injection of pure water has been of benefit.

The *prognosis* of morphinism as regards cure is not so favourable as was at one time believed.

Several considerations influence the result.

(1) The duration of the habit. Those cases which have lasted only a few months are more easily treated, and with greater certainty against relapse, than those which have gone on for several years, subject to the deleterious influence both on body and mind.

This requires no further explanation.

(2) When the condition which originated the use of morphia continues, or is incurable, a cure of morphinism can scarcely be looked for.

(3) The physical, but more especially the nervous, constitution plays an important part. Very weakly individuals fall into such a state of prostration on withdrawal of their accustomed drug, that it is necessary to give it again. In other cases where there is a strong predisposition to nervous disorder, the withdrawal of the drug gives rise to intense psychological disturbances.

(4) The maximum dose taken daily is not of great importance in relation to prognosis. The tolerance of different individuals varies so much, that what is a large dose for one is only a moderate dose for another.

Attention to these points may indicate what are the prospects of cure in a given case, but by no means with certainty. On the contrary, many morphinists go away apparently cured, and full of gratitude to the physician who has freed them from their misery; yet, as the subsequent history shows, the cure has been merely apparent. On this more hereafter.

Cases are sometimes met with in which the symptoms consequent on withdrawal of the drug are so serious as to necessitate its re-administration. As a rule, however, these symptoms should not be allowed to become too intense, and an allowance of a smaller quantity of the necessary drug enables us to dismiss such a patient, at least better, though not cured. In the great majority of cases, however, it is possible to withdraw the drug entirely; but then the question

arises—with what benefit? In those cases which, according to the points mentioned above, allow of a favourable prognosis, it is frequently possible to effect a permanent cure; but if all the conditions are not fulfilled, the prospect in my experience is distinctly gloomy. Very often a relapse occurs after a short interval, and the individual returns to his old ways. In the worst cases, mental disorders, frequently characterised by diverse hallucinations, but more especially of a melancholic form, with pronounced suicidal tendencies, are apt to ensue. In reference to relapses, the frequency of which Erlenmeyer ('*Centralblatt für Nervenheilkunde*,' No. 22, 1879) has recently noticed, I may mention the case of a medical man who, after four attempts at weaning, was treated in our institution, and dismissed apparently well, and who yet after three months was again in his former state. Many of the patients from whom the drug has been entirely kept for a length of time fall into a state of apathy, mental incapacity, and purposelessness, still retaining those moral characteristics already described. Among the eight cases described below there are three in which the results were suicide by gunshot in two, and in the third—a lady—repeated and most determined attempts to make away with herself. Hence the physician who undertakes to wean a morphinist of his habit must be very careful both as to his prognosis, and as to his means of carrying out the treatment to its end. Not unfrequently the patient will be most benefited by a diminution of his quantity, while a complete withdrawal will produce only other disorders. Very strict supervision of the patient for some months after active treatment is also highly desirable, though not always easily carried out. Indeed, in many cases in which there is physical or other discomfort rendering life otherwise unendurable, it is better to continue the administration of morphia, than interfere with the habit. The following example will illustrate this.

A medical man, aged 52, a Pole, was at the end of 1878 in a great state of alarm and fear of infection by the plague, so that he could no longer attend to his work, and became depressed and lachrymose. In spite of various remedies, such as bromide of potassium, chloral, &c., his symptoms increased;

he had intense precordial anxiety and sleeplessness, tore his hair, and felt inclined to commit suicide. On admission into our institution on May 20, 1879, he was in a state of great excitement, weeping, howling, and, while detailing his woes, would suddenly start as if terrified at something.

This condition continued the whole day, till 8 in the evening. At this time he became quiet and spoke rationally, exhibited some appetite, and felt quite well. Next day, however, the old condition returned. After various means had been employed, morphia injections were had recourse to. Twice or thrice a day, as seemed necessary, 0·01–0·02 gramme was injected subcutaneously. The effect was very marked. He became quieter and more hopeful, and gradually the quantity of morphia could be diminished. On July 31 he left the institution, but remained under supervision in the neighbourhood for three months longer. By the continued use of about 0·02 gr. morphia daily, he was more cheerful and contented, his weight increased, and his general appearance improved. His love for, and ability to work, however, have not yet come back.

Morphinism may be of medico-legal interest. The moral degradation which accompanies morphinism, like alcoholism, may lead to actions which bring the individual into collision with the law. It is not likely, however, that such a condition would be considered as a ground of irresponsibility. On the other hand, a case of simulation of morphinism lately occurred in Vienna. A young man was apprehended in the act of picking the pockets of two ladies at the same instant in a crowd. He excused himself on the ground of being a morphinist, and that through this "his cerebral blood-vessels were so congested that his ideas of things were perverted." He offered to take 1·0 gramme morphia, at one dose, in order to show, by his bearing it, that he was a morphinist. In the course of further observation by the police surgeon it became clear, however, that the accused could not simulate the phenomena of morphia-hunger; and that the plea was entirely false, and put forward merely to screen a case of wilful theft.

A series of experiments were made on animals in order to study the phenomena of morphinism in them, but though I

have carried on experiments for four months, I have not obtained any very characteristic results. I injected daily in a number of rabbits a quantity of hydrochlorate of morphia, beginning with 0·015–0·02 gramme, gradually increasing up to 0·3–0·4 gramme.

First it was observable that a similar habituation occurred in rabbits as in man. The animals treated by a gradually increased dose bore large doses of the drug much better than others. The symptoms observable after long-continued injection were, a certain want of tone and weakness of the muscles, most marked on the hinder extremities; and also trophic affections of the skin. In many of the animals the hair began to fall out, particularly on the neck, so that the skin became quite denuded in patches.

I could not make out any permanent contraction of the pupils; the vessels of the ears were shortly after the injection contracted, but later became dilated. In several animals, in order to ascertain if anything like morphia-hunger occurred, after three months daily injection—amounting at last to as much as 0·3–0·4 gramme—the administration was suddenly stopped. I cannot, however, say that I noticed any symptoms worthy of note. Perhaps one or more exhibited a slightly-increased reflex excitability, but within very narrow limits.

Owing to the comparative unimportance of the results obtained, I have not thought it necessary to record the experiments in detail. I will only mention that many rabbits, at varying periods in the course of the experimentation, i.e. in the first few days, or after several weeks, &c., died somewhat suddenly in convulsions. On section, the brain was found oedematous and swollen, while the other organs presented no discoverable abnormal appearances. Where such were visible, they were certainly not attributable to the morphia.

In conclusion, I append a few cases of chronic morphinism, which have been treated in my institution, with the remark that the first three cases have been already alluded to by Leidesdorf in his valuable work.

CASE 1. C——, aged 35, formerly lusty and cheerful, had, a year before his admission, taken to morphia in consequence of acute periostitis. He rapidly increased the dose, and on



many days he had taken 80 injections of 0·02 gramme each. As the effect of the injections, after which he felt fresh and strong, wore off, and the more rapidly each time while the reaction became more intense, he resolved to place himself under treatment, in order to wean himself entirely. With this view he was treated by an able physician, and with success. But immediately after the weaning, he fell into a condition of melancholia, which rapidly increased. He fancied himself a wretch, who had ruined the whole world; felt in a constant whirl, and could give no account of what passed before him. His memory was very defective; he was restless, particularly at night, when black thoughts drove him from his bed. On the journey he made a suicidal attempt, inflicting a wound two inches long on his throat with a razor, with the purpose, according to his own statement, of relieving the congestion of his head. He was brought to our institution. His symptoms improved so rapidly, that after six days his wife insisted, contrary to medical advice, on removing him. We learnt that a few months afterwards he had shot himself in the Tyrol.

CASE 2. S—, aged 33, had begun morphia injections seven years before admission, in consequence of injury of the sciatic nerve. Though the pain had speedily subsided, he had continued the morphia, and had gone on increasing the dose up to 2·0 gramme daily. Numerous abscesses resulting from the injections compelled him latterly to take the morphia chiefly by the mouth. He imported this direct from England in large original bottles, containing 100 grammes, and he took it with a spoon without dissolving it. The quantity which he took in this way must have amounted to nearly 3 grammes daily. Several attempts to wean himself having failed, he placed himself under our care. He had a pale-greyish complexion, and flabby muscles; but he was otherwise well nourished. He complained of apathy, and bad memory. He received on the first day 2·0 grammes, then 1·5, then 1·0, and on the fourth day 0·5 gramme morphia; while on the sixth it was entirely suspended. At the same time warm baths, strong wine, and at night beer was prescribed. Already at the diminution of the dose he was restless, complained of alternations of heat and cold, diarrhoea, prostration; and he could

obtain no sleep, even with chloral hydrate. Four days after the discontinuance of the morphia, a condition of collapse came on, so that it became necessary to give him an injection of 1·0 gramme. After this, and under the influence of bromide of potassium, the symptoms slowly improved, the appetite returned, but sleep for a long time was very poor. After eight weeks he was discharged; but according to later accounts he continued unable to turn himself to any employment, though he kept from morphia.

CASE 3. R—, aged 30, is the case already mentioned (p. 451) of the physician who became a morphinist from experimenting on himself with regard to morphia-hunger. At first he took very moderate quantities, but later, becoming acquainted with another medical man also a morphinist, he increased his allowance. This other medical man also came under my treatment. (Case 4.)

The reason of R.'s coming to the institution was neither the inconvenience of the abscesses caused by the repeated injections in the thighs, nor the costliness of the drug, but, as he truly said, owing to a moral "Katzenjammer:" a feeling that he was not as other men, but had artificially to be kept up to the level. Before he came under my treatment he had four times tried to wean himself, but each time unsuccessfully. First, voluntarily, while he was still attached to the hospital; second, involuntarily, when he was unable to return from a journey and had to remain two days without a supply; third, at a hydropathic establishment in the Steiermark; and lastly, at a vegetarian institution in Switzerland. Latterly he had been in the habit of injecting about 2·0 grammes daily. On one or two occasions he happened to puncture a vein with his syringe, so that the morphia was injected directly into the blood. A strange itching sensation immediately overspread the whole body, and great congestion and a feeling of bursting of the head came on, with red, cyanotic face, giddiness, and laboured breathing. In the course of from 2–10 minutes after the injection the symptoms generally subsided. The quantity was diminished gradually each day, so that on the 8th he received none. The phenomena of morphia-hunger then showed themselves in all their intensity. He was highly

excited, threatened to make away with himself, declared that he was illegally detained, and swore he would call the police to his assistance. He broke the furniture; ate almost nothing for some days; complained much of cold, especially towards evening when he went to bed; violent diarrhoea; the pulse, which was formerly usually 72, sinking to 54. Then occurred hyperæsthesia and paræsthesia of the various senses; everything smelt and tasted bad. He was ordered bromide of potassium, wine, and warm baths. Gradually he became more quiet; his strength improved, but he continued for a long time very irritable and pugnacious. His pulse rose to 100. He began again to smoke and to seek amusement. He went away contented and happy and full of gratitude, after four weeks' stay in the institution. The first accounts were favourable enough, but half a year later he had already gone back to his old ways, and was as bad as ever.

CASE 4. H——, aged 32, a medical man, had taken morphia for five years, having commenced it in consequence of tooth-ache. As he found the morphia relieve also some mental troubles and worries, he continued with it. He gave also as another reason that, being assistant at the Physiological laboratory, he saw in himself an interesting subject of experiment. He found the internal administration better than the subcutaneous, though the action was less rapid. Various attempts to give up the habit failed. He took daily 1·2–1·3 grammes, and had also induced Case 3, whom he recognised as a morphinist by his aspect, to indulge more deeply than he had been wont.

The symptoms which he chiefly complained of were copious perspirations and pharyngeal catarrh. The ashy-grey complexion, peculiar to morphinists, was in him exceedingly well marked.

For the first few days of his stay in the institution he received from 0·6–0·2 gramme daily. On the sixth day it was entirely suspended. Then the phenomena of abstinence exhibited themselves in an intense degree. He was very excited, and wanted to climb up the wall with his feet. He had violent sneezing, sweating, and diarrhoea. Sacral pain; a feeling of coldness, chiefly one-sided; yawning, formication in

every part of the body, were the chief symptoms, in addition to great prostration. It was noticed that he very soon exhibited a profound aversion to morphia.

The treatment was similar to that of the former cases. Cherry-laurel water was very useful in calming his restlessness. His sleep was at first very indifferent, but improved under morphia, though he affirmed that he did not sleep at all, which was not really the case. His complexion became again fresh and ruddy. His desire for life and enjoyment returned, and, contrary to our advice, he went to the theatre on the 18th day, and left the institution at the end of three weeks apparently quite cured. He went to Italy, and returned after two months cheerful and happy at the successful cure which had been effected. He stated that during his sojourn in Italy, being attacked with diarrhoea, he had taken thirty drops of opium, a dose which he had found sufficient during his morphinism. This, however, was said to have produced symptoms of poisoning. He took up his residence in Vienna, but shot himself fourteen days afterwards. Shortly before, as we learnt on inquiry, he had on several occasions taken large doses of morphia.

CASE 5. R—, aged 47, farmer. The patient's father was insane. The patient himself was formerly subject to headaches. Nine months before his admission into our institution, in consequence of overwork and worry, he began to suffer with gastric catarrh, palpitation and despondency. He could no longer maintain his family, could not work, was sleepless, and had creeping sensations all over his body, as if all the ants in the world had been let loose on him. All remedies proved of no avail; chloral was useless; morphia never relieved him bodily and mentally. He injected daily 0·15–0·02 gramme. Under hydropathic treatment in Gräfenberg he improved still further, but he was advised also to give up morphia entirely before he could be considered cured. Under the existing conditions, however, a weaning process seemed neither necessary nor likely to succeed, but at the very earnest desire of R. himself he was admitted as a patient. During the last few days he had taken what for him was a considerable dose daily, viz. 0·35 gramme, and had the symptoms of acute

morphia-poisoning on his admission—constipation, giddiness, somnolence, hallucinations of hearing. For the first twenty days of his stay he received morphia injections of gradually diminished amount, yet he very soon began to exhibit symptoms of morphia-hunger—formication, cold, diarrhœa, restlessness; and all the symptoms for which he took morphia came back. He complained of terrible dreams, and when in bed kept his legs in constant motion. The symptoms showed themselves with special intensity when the morphia was completely suspended. The psychical symptoms chiefly predominated. He threatened to prosecute everybody, beat his attendant, looked very wild, swore he must die, could not endure life longer. A violent pain extended from his hypogastrium through his limbs, and he complained also of pain in his joints and on both sides of his head. By means of chloral and bromide of potassium, administered chiefly as enemata, sleep could usually be induced. During the ten days of his abstinence the symptoms rather increased than diminished; he refused to obey instructions, and became ultimately so weak and unmanageable that morphia had to be again allowed. At first he received 0·15 gramme twice a-day; but as this did not prove sufficient, it was increased to 0·09 gramme daily, given in three doses. Hereupon the psychical symptoms disappeared, with the exception of slight hypochondriasis; and his bodily condition improved, particularly his gastric catarrh. He left the institution, but returned after his daily dose had reached 0·18 gramme, which was in the course of three weeks. He did not, however, enter the institution, but lived in private under the care of my assistant, Dr. Krueg. Under his treatment the quantity was reduced to 0·09 gramme daily, and with this the patient continued free from his troublesome symptoms, and returned home.

CASE 6. M——, aged 33, a medical practitioner, had begun morphia seven years before, in consequence of periostitis of the lower jaw. He had, however, ceased taking it, but recommenced on being attacked by pleurisy, and had gone on, till lately he was in the habit of injecting as much as 0·6 gramme daily. He noticed, however, that he became less fit for work, that his energy failed, and therefore he resolved to

wean himself. On his admission the morphia was entirely suspended at once. For the first two days of abstinence he felt quite well, but sneezed a great deal. In the night of the third day he was seized with violent vomiting, repeated about 80 times within the 24 hours. The quantity of vomited matters was enormous, so that in this one day he became very low and prostrate, felt cold, and complained of sacral pains. The pulse, which at first was 76, sank to 46. Next day the vomiting ceased, the pulse was 42; there was great prostration, with diarrhoea. The colour of the stools was, as it is generally, very dark, almost black. From this time he recovered rapidly, so that already on the eleventh day he left the institution. His pulse was then 90. As to his subsequent history I have heard nothing.

CASE 7. Frau S—— had taken morphia for two years, in consequence of a painful abdominal affection, and had come to taking 0·2 gramme daily. Under the morphinism her general health was deteriorating so much that she resolved to place herself under treatment for the purpose of being weaned. For the first two days a little morphia was allowed, but she already at the diminution of the dose complained of pain in the sacrum, general malaise, tremor of the limbs, creeping sensations all over, cold, dislike to food. She was very restless, yawned a great deal and could not sleep. These symptoms became more intense on the third day, when the drug was entirely suspended, but after two days, improvement occurred. Lactate of soda (10 grammes) once procured a few hours' sleep, but on the following nights proved of no use. For a few days she continued very irritable and excitable, but was dismissed as cured in a fortnight. She made a tour to Italy, and returned at the end of six months, quite fresh and blooming.

CASE 8. Frau S——, a highly intellectual lady of nervous constitution, had been troubled for several years with nervous vomiting, which occurred particularly in the morning; and also with intense headaches. Of all remedies, morphia gave the greatest relief. Taken internally, it had scarcely any effect, but was active when injected subcutaneously. She stated that during the injection she often felt the blood

rush to the head, and that she often experienced a peculiar metallic taste in the mouth, which increased during the first 40-50 seconds, and then gradually faded away. Gradually she raised her allowance to about 2·0 grammes daily. She had made repeated attempts to wean herself, but unsuccessfully, as the above-mentioned nervous symptoms returned. She affirmed she even had an aversion to morphia, but could not do without it for the reasons mentioned. When she had reduced herself to 0·02 gramme daily, she suffered so much that she was obliged, contrary to her desire, to increase the dose again.

From this habit she had acquired a sodden sallow complexion, and a peculiar look of illness about the eyes. It is to be noted that she herself said that no one should trust a morphinist, as they were all alike untruthful. Such was the condition of things six months before her admission into the institution. Shortly after that she had again attempted to conquer the morphia, and had employed large doses of chloral against her sleeplessness. When the morphia was entirely suspended, dangerous nervous symptoms supervened. She was very excited; was often delirious; talked verses for hours; had tetaniform attacks with such intense prostration, that she was sometimes thought dead; had pains in every part of the body. Frequently she lost consciousness, but at other times she had intervals of perfect rest and clear-headedness, during which she earnestly requested to be taken to the institution.

During one of these lucid intervals she was brought to the institution on Dec. 7, 1877, in the evening. She spoke quite intelligently and sensibly about her condition, but within three hours she had a violent maniacal paroxysm. She dashed about the room, screamed, struck out, and could scarcely be held by four men. She was at the same time filthy in her habits. Next day she was quieter, but was frequently unconscious, and did not know where she was. In the course of December these attacks of excitement occurred repeatedly. At the same time her intelligence was impaired, her appetite bad, and large doses of chloral scarcely procured an hour's sleep. The urinary secretion was much diminished. She wasted considerably. Towards the beginning of 1878, during the first weeks

of the new year, she began to be quieter and gain strength. With this her mental condition quite changed. She was clearer, and generally recognised people; but she was highly crazed, had many hallucinations:—everything stank; there was a corpse in the room. She made repeated attempts at suicide; tried to set fire to her dress, and to strangle herself. After several weeks a state of apathy came on, which lasted till April. She scarcely spoke a word; frequently, however, she declaimed, lay almost constantly in bed, and took no interest in anything around her. In April she was again more active, but talked utter nonsense: “I will to the everlasting police; I have devoured my husband!” When out walking, she several times attempted suicide, trying to jump into a water cask. In May she was removed to another institution, which she left cured in May of this year (1879).

I saw her two months later. She was looking well, her mind had its normal clearness, and she said that she remembered scarcely anything of my institution. She remembered only one or two special circumstances.

In conclusion, I give the tables of the more important papers on Morphinism, so far as they are known to me.

*Bernhuber*.—Aertz. Intelligenzblatt, 1878.

*Burkhardt*.—Die Chronische Morphinium-Vergiftung. Bonn, 1877.

*Busey*.—Philadelphia Medical Times, 1876.

*Calvet*.—Essai sur le Morphinisme. Thèse de Paris, 1876.

*Erlenmeyer*.—Die Recidive der Morphinium-Sucht. Centralbl. f. Nervenheilkunde, 1879.

*Fiedler*.—Ueber den Missbrauch der Morphin-Injectionen. Zeitsch. für prakt. Medicin, 1874.

*Kormann*.—Deutsche med. Wochenschrift, 1877.

*Krage*.—Ueber Albuminuria und Glycosuria nach Morphinium. Greifswald, 1878.

*Kunz*.—Die Morphiniumsucht. Baier. Aertz. Intelligenzblatt, 1876.

*Laehr*.—Ueber Missbrauch mit Morphin-Injectionen. All. Zeitsch. für Psychiatrie, 1872.

*Leidesdorf*.—Die Morphiniumsucht. Wien. med. Wochenschrift, 1876.



*Levinstein.*—Several papers in the Berl. klin. Wochenschrift, 1875, *et seq.* Die Morphiumsucht. Berlin, 1877.

*Lewin.*—Ueber Morphiumintoxicationen. Zeitsch. f. prakt. Medicin, 1874.

*Martin.*—The Opium Habit. Philad. Med. Times, 1874.

*Mattison.*—Philadelphia Med. and Surg. Rep. 1874. New York Med. Record, 1876.

*Michel.*—Ueber Morphiuminjectionen und Morphinismus. Würtemb. med. Correspondenzblatt, 1876.

*Moinet.*—On the Administration of Opium. Edin. Med. Journal, 1875.

*Richter.*—Casuistik zum Morphinismus. Berl. klin. Wochenschrift, 1876.

## ILLUSTRATIONS OF DISEASES OF THE PONS VAROLII.

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THE special symptoms of acute lesions of the Pons Varolii may be briefly epitomised as follows :

1. The involvement, in unilateral lesions, of cranial nerves on the same side as the lesion.

2. The frequency of bilateral symptoms of paralysis in the cranial nerves and limbs. This depends on the fact that the motor tracts from the two sides are here still in contiguity, and on the circumstance that both are supplied from one vessel, the basilar artery, which is an occasional seat of morbid processes ; degeneration, syphilitic disease, thrombosis, embolism.

3. The implication of the centres which are situated at either extremity of the pons, and which by their irritation and paralysis give rise to important localising symptoms. In the upper portion of the pons is the centre of origin of the oculo-motor nerves, irritation of which gives rise to the well-known contraction of the pupils, and damage to which may cause an equally important paralysis of the irides and ocular muscles. At the lower extremity of the pons are the centres which influence respiration, deglutition, convulsion, &c. Lesions which destroy these rarely leave time for diagnosis, but their irritation furnishes us with most important diagnostic indications.

4. The occurrence of hyperpyrexia, which appears to ensue from acute bilateral lesions irrespective of their character.

5. The occurrence of glycosuria, which probably is found only after the acute process is over, and has, in diagnosis, only a corroborative value.

Many of these symptoms are illustrated by the following examples of diseases of the pons Varolii,—embolism of the basilar artery, hæmorrhage, and senile softening.

CASE 1.—*Embolism of the basilar artery; general paralysis; myosis; hyperpyrexia.*

Wm. G., æt. 48, labourer, admitted into University College Hospital, Feb. 9, 1877. Rheumatic fever at the age of 28. For six months before admission he had suffered from shortness of breath, and for some weeks from bronchitis. He was a stout plethoric-looking man, presenting considerable dyspnœa, slight cyanosis, and œdema of the legs. Temperature normal. His lungs were emphysematous, and there were abundant bronchitic râles. Pulse 88, almost imperceptible, irregular and intermittent. Heart's action tumultuous; impulse diffused; dulness obscured by emphysema. First sound, rough; no murmur. Urine, sp. gr. 1024–30; albumen,  $\frac{1}{4}$ . Numerous hyaline, granular, and epitheliated casts.

On admission it was noted that the right pupil was larger than the left, both acting, however, to light. Two days after admission (on the 11th) he had a very severe attack of dyspnœa, without apparent cause. In the evening his pupils were contracted, but equal, and acting slightly to light. Patient was restless, wandering slightly; answering, however, when spoken to. This state continued during the night. At 7 A.M., on the 12th, he sank into the following condition. He lay on his back, breathing stertorously, skin hot, covered with perspiration. Pupils much contracted, equal, and insensible to light. He did not answer when spoken to, but could be slightly roused, opening his eyes when addressed in a loud voice. Eyelids habitually closed. Limbs not moved, and somewhat rigid; when pinched, an attempt was made to draw them away, but this seemed rather like a reflex than a voluntary action. At 10 A.M. his condition was nearly the same: when spoken to he still moved his head slightly and opened his eyes. Ophthalmoscopic appearances normal.

This state continued throughout the day. Early the next morning (3 A.M.) the coma had become deeper, and no sign of recognition could be elicited when patient was spoken to.

Pinching the arms still produced a slight movement. Pupils were contracted to a pin's point, and did not act at all to light. Swallowing impossible; temperature,  $105\cdot6$ ; pulse, 130; respirations, 17. At 5 A.M. the temperature had risen to  $109\cdot2$ . A quarter of an hour later he was seized with a fit of coughing, became more cyanosed, and died in a few minutes.

The albumen in the urine lessened progressively from  $\frac{1}{4}$  on the day of admission, to  $\frac{1}{20}$ , about, on the day before death.

*Neeropsy* (30 hours after death).—Heart very large, weighing 23 oz. All parts flabby and dilated; the left ventricle especially, its wall scarcely  $\frac{1}{2}$  inch in thickness. Orifices all dilated: mitral,  $4\frac{1}{2}$  inches; aortic,  $3\frac{1}{2}$  inches; tricuspid,  $5\frac{1}{2}$  inches; pulmonary, 3 inches. A quantity of old membranous clot in the left ventricle, partly interlaced among the columnæ carneæ. Left auricular appendix distended with clot; from it there projected a mass of old clot, the size of a walnut, which had firm adhesions to the wall in front of the appendix. The muscular fibres of the wall of the left ventricle were very granular.

*Kidneys* large: left, 9 oz.; surface studded with small cysts; much fat in hilus; cortex narrow, consistence lessened; section opaque. Right: 7 oz., fewer cysts, firmer; cortex enlarged; epithelial cells very granular.

*Liver and lungs* congested.

*Brain*.—No lesion above the pons; arteries at the base fairly healthy. Vertebrales and basilar free from atheroma. The anterior half of the basilar distended with clot, reaching forwards as far as the anterior cerebellar arteries. The anterior portion of the clot was pale, and distended the vessel more than the other part of the clot, which was dark, recently-formed coagulum. The anterior portion was evidently an embolon, and resembled perfectly the old clot in the left auricle. Both superior cerebellar arteries contained fluid blood, except that the first quarter of an inch of the right contained a coagulum continuous with that in the basilar. Both posterior cerebrals also contained fluid blood, although clot extended into the commencement of the right, which was very much smaller than the left—about one-fourth of the size—the right posterior communicating artery being correspondingly large. The pos-

terior half of the basilar, and the vertebrals, contained fluid blood.

The lower half of the floor of the fourth ventricle presented no vascular engorgement, only one or two vessels of the smallest size being visible. In the upper half a large vessel on each side was enormously distended with blood, and the grey matter had a redder tint than normal. The consistence of the upper half of the pons was slightly diminished, but there was no actual softening. (Being intended for a museum preparation it was not further examined.) The cerebellum was healthy.

The case was a typical one of embolism of the basilar general paralysis, with persistence of quasi-voluntary reflex movements, peculiar incomplete coma, irritation of the nucleus of the third nerve (or pupillary centre) leading to the extreme myosis, and with hyperpyrexia.

It is remarkable that some authorities doubt the occurrence of embolism of the basilar artery. Nothnagel, in his recent most admirable work on cerebral localisation,<sup>1</sup> says, "Acute embolisms of the basilar artery, limited in effect to the pons, do not occur. The reason for this is clear, on consideration of the anatomical relations of the vascular arrangement of the vertebral arteries, which renders the entrance of a plug into them extremely difficult, and on consideration that these vessels have a narrower lumen than the basilar artery which arises from them. If thus a plug should enter the vertebral artery, it must remain arrested therein, and it can, at most, cause only a gradual thrombotic occlusion of the basilar by the deposit of clot."

This reasoning is, I think, scarcely conclusive. The passage of a plug into a vertebral does undoubtedly occur now and then. The posterior cerebral artery may be plugged by an embolon. The difference in size between the vertebral and posterior cerebral arteries is considerable. A plug may be small enough to pass through the vertebral and yet too large to pass into the posterior cerebral. In this case it must be arrested at the anterior extremity of the basilar,

<sup>1</sup> 'Topische Diagnostik der Gehirnkrankheiten:' Berlin, 1879.

and whether its effects are limited to the pons or not will depend on the size of the posterior communicating arteries. As regards embolic processes, the basilar is an indifferent continuation of either vertebral, and the anterior portion of the basilar is the necessary place of arrest for a clot just small enough to pass through the vertebral. Probably Nothnagel's opinion is intended to refer to plugging of the course of the basilar, not of its anterior extremity. But many embolic plugs are much longer, in their longest dimension, than is the transverse diameter of arteries through which they pass. Such a plug may be washed along the vertebral, and plug a considerable extent of the anterior portion of the basilar, and even under the pressure of the blood behind may distend the vessel so as to give the impression of a plug which could not have passed through the vertebral.

Thrombosis in the basilar artery, the consequence of arterial disease, atheromatous or syphilitic, is however unquestionably a more frequent lesion than embolism. It probably existed in a case (seen with Dr. Vereker Bindon) in which the disturbance of respiration was very striking. The patient was a stout woman, aged 56, who had had several slight rheumatic or gouty attacks. After exposure to cold she became ill, complaining of sciatic and lumbar pains, and some left-sided pleurisy was found three days later. Her urine was free from albumen. In the afternoon she complained of ringing in the ears, suddenly became slightly delirious, and then partially unconscious; pupils slightly contracted, equal, and still sensitive. Respiration became embarrassed, without anything being discoverable in the chest to account for it, the breathing, at intervals of about twenty minutes, becoming slower and slower, until it ceased; but on artificial respiration being adopted, it gradually recommenced and gained in strength for about ten minutes, when it again went through the same process. At 10 P.M. she was lying on her back, moving the head uneasily from side to side, and at times opening her eyes, but not the least evidence of consciousness could be elicited. The pupils were of medium size, equal, not acting on the least to light. The arms were flexed at the elbow and rigid, with constant

jerking, half-co-ordinated, but evidently involuntary, movements of the hands. The legs were motionless. Respiration was very irregular, frequently stopping for a few seconds and then going again. Skin perspiring, temperature  $102^{\circ}$ . Pulse 120. The intermitting respiration continued through the night and next morning became extremely irregular, often ceasing for a minute at a time. She died next day at noon. There was no post-mortem.

In this case the bilateral symptoms, the convulsive movements in the arms, the inaction of the pupils, in contrast with the apparently incomplete degree of coma, the initial tinnitus, and lastly the extreme disturbance of respiration, point strongly to a lesion of the pons, probably thrombosis of the basilar artery, due to the blood state.

Degeneration of the basilar artery usually produces its effects by leading to thrombosis within it, but may lead to fatal results without this, by obliterating the branches which are given off to supply the pons. This occurred in a patient under my care several years ago, of whose case I possess only a very brief record. He was a man about 50 years of age, who had had a transient attack of left hemiplegia six months before he came under treatment, and two weeks before being seen he had a fresh attack, also on the left side, coming on without loss of consciousness, and reaching its maximum in three days. On admission into hospital there was complete paralysis of the left arm and leg, and moderate paralysis of the face without loss of sensation. Two days subsequently the right arm and leg also became weak, and the weakness increased during the next two days. Respiration became frequent (45 per minute), the chest full of râles, the skin perspiring. He died three days after the commencement of the right-sided weakness. His skin before death was said to have become extremely hot. The post-mortem revealed no lesion in the hemispheres. The basilar artery was extremely atheromatous, and there was an area of softening on the right side of the pons near the middle.

In another case of thrombosis in the basilar from syphilitic disease, lately under my observation, the temperature rose before death to  $105^{\circ}$ . A similar temperature was observed in the next case of rapidly fatal hæmorrhage into the pons.

CASE 2.—*Hæmorrhage into the Pons Varolii: oculo-plegia; respiratory disturbance; convulsion in legs; hyperpyrexia; death within an hour.*

A man, apparently about 60 years of age, was brought to University College Hospital about a quarter of an hour after he had fallen unconscious in a public-house. He then presented no sign of consciousness, eyes partly open, pupils medium size and absolutely insensible to light. Conjunctiva insensitive to touch. Pulse weak and regular, not infrequent. Breathing was not stertorous, but was markedly irregular and interrupted: three or four respirations were taken, and then a long pause ensued, during which there was some venous congestion of the face. At the end of each breathing period, when the chest was full of air, there was a forcible respiration, very like a sneeze, which jerked his head off the couch for some inches. Then followed the pause. The arms were motionless and flaccid. The lower limbs and muscles of the abdomen were affected with "convulsive tremblings."

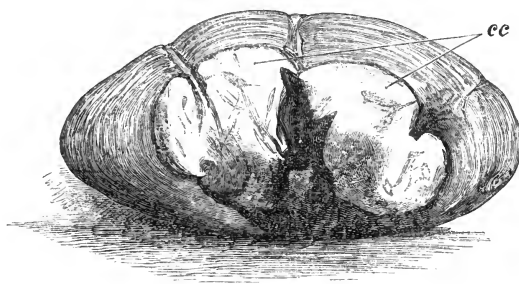
I saw the patient immediately after his admission into the ward. The peculiar sneeze-like spasm of respiration had ceased: his breathing being still very irregular. The convulsive movements in the legs continued—constant, quick, jerking movements, equal and synchronous in the two. After about half an hour the arms also became rigid and convulsed. His temperature, when first seen, appeared normal, but soon after admission into the ward it was  $103^{\circ}$ . He died, asphyxiated, three-quarters of an hour after admission, and about an hour after the seizure. At death the temperature was  $105^{\circ}\cdot4$ .

At the necropsy a large clot was found to occupy the pons Varolii, being most extensive in its upper and anterior portion, the upper (posterior) half of the pons being destroyed and the anterior (inferior) portion considerably damaged. The clot had burst through into the fourth ventricle, which was filled with blood—the upper half of the floor being completely broken up. The destruction ceased below at the auditory striæ. It extended to the surface in the middle of the pons, separating the superficial fibres midway between the right fifth and sixth nerves. It extended upwards beneath the corpora quadrigemina,



where it reached backwards along the middle line almost to the surface, completely destroying the nuclei of the third nerves. The inner side of the left crus was torn by the

Fig. 1.



Section through the crura cerebri (*cc*) just above the pons: the space between them at the upper part (below in the figure) is occupied by hæmorrhage, which extends almost to the under surface of the pons (above in the figure), and has extensively damaged the corpora quadrigemina and destroyed the nuclei of the third nerves. (*From a sketch by Mr. Neale.*)

hæmorrhage, and the right crus in less extent. The hæmorrhage had extended into the right crus cerebelli, but not quite so far as the corpus dentatum. The third ventricle was filled with clot, and there was fluid blood in each lateral ventricle. There was a little blood at the base beneath the arachnoid. Very little atheroma of the vessels.

The left lung was intensely congested. The lower lobe appeared to be a mass of blood. The substance of the lung was black, but of normal consistence, and crepitant. The upper lobe was loaded with fluid but not congested. The right lung showed only some old changes. The left kidney was enlarged and slightly indurated: the right atrophied, in consequence of an impacted calculus in the pelvis, which had caused hydronephrosis. In the stomach, over the left third, the mucous membrane was covered with extravasations, small, recent, arranged in an arborescent form.

The case illustrates many points of interest in the diagnosis of these diseases. The nuclei of the nerves moving the eyeball and the pupil were destroyed. Hence both eyeballs and pupils were motionless, and the pin-point con-

traction, which exists when the lesion merely irritates the nuclei of the third, was absent.

The respiratory centre situated just below the lesion was irritated and disturbed, and hence the remarkable respiratory symptoms.

The elevation of temperature in disease in this situation is of interest in its diagnostic as well as its physiological relations. The diagnosis of hæmorrhage into the pons was doubted, at the bedside, by some of the students, on the ground that hæmorrhage causes an initial depression of temperature in all cases. In some authorities the statement is certainly made too absolutely; the fact of the special effect of lesions on the temperature of the pons, which is independent of the form of lesion, being overlooked.

The extreme congestion of the left lung and the extravasations in the left end of the stomach may possibly have been due to the irritation of the nucleus of the pneumogastric.

CASE 3.—*Softening of the Pons Varolii; hemiplegia; subsequently opposed facial palsy; diplegic spasm; glycosuria.*

Robert J., five months before being seen, was attacked with right hemiplegia: there remained considerable weakness of the right arm and right leg: very little paralysis of the right side of the face. On December 13th, four months after the onset of the hemiplegia, after riding in a wet cab, he felt pain in the left jaw, which became so intense as to prevent him from sleeping: it radiated from behind and below the left ear over the side and front of the head, and over the left side of the face. The intense pain lasted three or four hours. On waking next morning the left side of the face was completely paralysed in all parts. The pain continued until he was seen on January 3rd. The paralysis of the face had improved slightly, but was still nearly complete. Faradic irritability 4 or 5 cm. (Stöhrer sec. current) less than right side; voltaic irritability four cells greater. The application of the voltaic current to the right side, especially to the point of exit of the second division of the fifth, or to the forehead, the other pole being placed in front of the ear, excited contractions in the muscles of the left side of the face, especially in the lev. labii sup., and to a less

extent in the zygomatici. They occurred on closing the circuit, and with very feeble currents (four cells). The same current which, passed from the ear to the forehead on the right side, excited a strong contraction in the muscles moving the angle of the mouth on the left side, caused no contraction in these muscles when applied to the left side of the chin. Further examination revealed distinct hyperæsthesia of the second branch of the right fifth nerve: pressure on, or pinching the skin causing much more uneasiness than on the left side.

On February 8th his urine, which had not been before examined, was found to contain a distinct trace of albumen, and a considerable quantity of sugar, ascertained next day to amount to 1800 grains in the twenty-four hours' urine, on the 12th to 2835 grains, and on the 14th to 1800 grains; the quantity of urine varying from 90 to 110 fl. oz. He was conscious of an increased flow of urine, and was confident that this had come on since the attack of hemiplegia. On strict diabetic diet the urine fell, in the course of a week, to 45 oz., and the sugar to a trace too small to estimate quantitatively. During the next two months the urine remained the same in quantity, and the trace of sugar persisted. The facial paralysis improved slowly. On May 12th he died suddenly—in a moment.

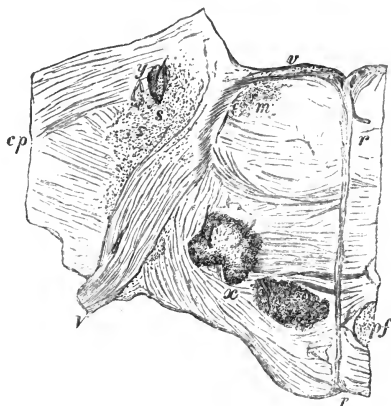
The necropsy showed a fusiform dilatation of the arch of the aorta, as far as the left subclavian, its lumen being about four times the normal. The heart healthy for age (11 oz.). The kidneys were large (8 and 11 oz.), opaque and indurated.

The brain above the pons Varolii showed no evidence of disease. The arteries of the base were very atheromatous, but nowhere occluded.

The pons Varolii, medulla, and spinal cord were carefully examined after hardening. The pons, just behind the origin of the crura cerebri, was equal in size on the two sides; but a little lower, opposite the upper portion of the middle peduncle of the cerebellum, there was a depression in the convexity of the left side; and opposite this, in the middle of the pons, was an irregular area of softening, situated in the pyramidal fibres, opposite and a little below the origin of the fifth nerve. Above, the softening was in two foci (*x*) (Fig. 2, p. 476), in one of which was a cavity, the other being occupied by

granular débris, but a little lower these two had blended into one of rather smaller size. The fibres of the fifth nerve and of the facial nerve were both apparently undamaged; the

Fig. 2.



Section through the left half of the pons Varolii at level of exit of fifth nerve: *v*, floor of fourth ventricle; *cp*, middle peduncle of cerebellum divided; *rr*, raphe of pons; *V*, fifth nerve at its exit; the fibres are seen ascending into the substance of the pons; *s*, upper part of the sensory nucleus, in which are two foci of softening, *y*; *m*, highest part of motor nucleus of fifth; *pf*, pyramidal fibres on right side ascending through the pons from anterior pyramid; *x*, two foci of softening, destroying all the ascending pyramidal fibres on the left side.

lesion, in its lower part, was close to the fibres of the facial. At the level figured, the sensory nucleus of the fifth (*s*) presented two small foci of softening (*y*). The anterior pyramid on that side was completely degenerated, and the degeneration could be traced through the decussation of the pyramids to the opposite lateral column of the medulla in the usual course.

*Remarks.*—The early notes of this case are imperfect, although the patient was very carefully examined, the symptoms present being those of disease of the corpus striatum with a small amount of paralysis of the face. The occurrence of facial paralysis led to a careful investigation with electricity, which revealed the peculiar diplegic contractions. From these disease of the pons was suspected, which the existence of glycosuria supported and the autopsy confirmed.

Although the autopsy did not reveal any actual lesion of the facial nerve within the pons, it seems highly probable that its paralysis was due to inflammation extending from the adjacent focus of softening, and that this extension and the lesion in the nucleus of the fifth occurred simultaneously. To the latter, the intense pain in the region supplied by the fifth may have been due.

The contractions in the left side of the face, when the right side was galvanised, can only have been produced by one of two modes. Either they were the result of direct stimulation of the very sensitive muscle by the irradiation of the voltaic current, or they were reflex. It seems impossible that they can have been due to the wide diffusion of the current. A current of four cells applied in the way described, could scarcely cause contractions in the muscles of the other side of the nose, especially when it is considered that a much stronger current, applied below the angle of the mouth on the left side, did not lead to the contraction of the levator labii superioris, &c., which followed the application to the right side.

The cause of the diplegic reflex was probably an undue sensitiveness of the fifth nerves, perhaps due to the lesion found in the nucleus of the left fifth, reacting on the right nucleus.

The occurrence of glycosuria in diseases of the pons which do not actually involve the "diabetic point" has been before observed. In this case the fact that the patient had observed the increased flow of urine only since the hemiplegia made it highly probable that the diabetes was a sequel to the paralysis. It is not necessary, in explaining the phenomenon, to assume that the focus of disease is related to the glycosuric processes. The vascular disturbance secondary to the obstruction which caused the softening would, no doubt, be very considerable, and may have extended to the "diabetic point."

## ON THE ORIGIN OF TETANUS.

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TETANUS is one of those few remaining, striking, and fatal diseases, which deeply interest the public mind, as having so long resisted the art of medicine, and baffled its inquiries. Not, indeed, that cases of tetanus are invariably fatal. From time to time well-authenticated cures are reported in the papers. But there is as yet no received theory as to the nature of the disease; there is no acknowledged line of treatment; and the number of deaths in acute cases is out of proportion greater than in most other diseases. Acute tetanus is even more fatal than cholera. Poland gives the mortality of all cases, as seven deaths in eight and a-half cases.

How comes it that we have advanced so little in this direction? Probably because there are still different, and even contradictory, views held and taught as to the very nature of the disease. There are humoral pathologists—a good number—who look upon tetanus as a species of blood-poison. Others trace it to inflammation of the nerves and spinal cord; while the majority, confronted with the dual origin of the disease, expressed by its classification into Traumatic or Idiopathic, consider, in fact, that it may arise either one way or the other. Of late, however, there is a growing tendency on the part of writers on tetanus to give up all of the foregoing theories as to the origin of the disease as lacking proof, and being inconsistent with many well-known facts. But, so far, nothing very definite has been put forward in substitution.

And first as to its humoral origin, which finds expression so often in the term Idiopathic, as distinct from the Traumatic

form of the disease. What proof have we of it? As a rule, perhaps, so-called idiopathic cases are milder than cases arising from injury; but the exceptions to this rule, both ways, are so numerous, that no argument can be founded on this. Many idiopathic cases are very acute; idiopathic tetanus is often fatal. Many traumatic cases, on the other hand, are very mild, and do not end fatally. The fact is that tetanus has grades of severity, like other diseases; acute cases, however arising, are generally fatal; and chronic cases, however arising, are generally not fatal; and the division into acute and chronic is a good and practical one, but there is no positive proof of its idiopathic origin in any single case.

It is easy to deny the humoral or idiopathic origin of tetanus. I must use the term idiopathic in this paper, to make myself clear, since it is a part of the language of the subject; but I do so with the protest that the word is in this connection a mere negative. It really means nothing more than that the cause of the disease is not apparently traumatic. It has, besides, the great practical drawback of suggesting a humoral origin where none has actually been found.

No one, as far as I know, denies that traumatism is one cause of this disease. There are so many unequivocal proofs of it on record, that it would be rash to do so. I will give one out of many for the sake of illustration, premising that the case is an unusual one. Sir T. Watson relates that Professor Robinson, of Edinburgh, was once at table when a negro servant lacerated his thumb by the fracture of a china dish. He was seized with convulsions almost instantly, and died with tetanic symptoms in a quarter of an hour. This case, it will readily be admitted, leaves no opening for humoral pathology. There are many cases which go nearly as far to demonstrate the traumatic origin of the disease. I think I may, to save space, take so much for granted, that tetanus does often arise from injury. It is generally so admitted, and all cases of tetanus following an injury are classed as traumatic. This leaves a large residue of cases to be dealt with, in which no injury is apparent, and which are for that reason called idiopathic, and for no better reason. I might here suggest that the traumatic origin of tetanus is often so insignificant as to be overlooked.

Sir T. Watson mentions cases of tetanus following the bite of a sparrow; the stroke of a whip-lash, in which the skin was not broken; a fish-bone in the fauces; a blow of a stick on the hand. Sir W. Ferguson, the scratch of a pin. Bryant, a slight contusion. Erichsen, a child falling on its back; no breach of surface. And doubtless some, so-called, idiopathic cases have grown out of some such trivial injuries. But the difficulty of explanation must be fully considered, and fairly met. What is the difficulty? That a number of cases of tetanus occur without the intervention of a wound or injury. And, what if the intervention of a surgical wound or injury be an accident of the disease, and not an essential? I am far from considering a surgical injury as the invariable starting-point of tetanus. On the contrary I would give up traumatic as well as idiopathic, looking upon both members of the classification as misleading. I will try to show that tetanus is a single disease; that it begins in persistent peripheral nerve-irritation, and ends in organic molecular, and functional disturbance of the medulla. This peripheral nerve-irritation need not, however, be traumatic.

I will divide the subject into five propositions. 1st. Peripheral nerve-irritation is a cause of tetanus. 2nd. Peripheral nerve-irritation is present in all cases of the disease. 3rd. It produces eventually the group of symptoms known as tetanus. 4th. It explains the facts of the morbid anatomy of the disease. 5th. It guides the treatment of the disease, and is proved by its success.

Three cases may be cited to prove that peripheral nerve-irritation is a cause of the disease. 1st. Dr. Murray, in the eleventh volume of the 'Medical Gazette,' records the case of a midshipman, who whilst on night-watch trod upon a rusty nail, which pierced his left foot. Soon after, his foot and leg felt numb and peculiar, and the next morning symptoms of acute tetanus set in. Dr. Murray at once divided the left posterior tibial nerve with a stroke of his scalpel. The patient felt immediate relief; his mouth opened; the disease subsided, and he was well in two or three days. Now the way in which I read this, and the only way in which I can understand it, is, that a peripheral branch of the left posterior tibial nerve was



injured, or irritated by the nail or wound, and that the irritation was propagated along the nerve to the medulla, and gave rise to tetanus; and that, upon the division of the nerve, the irritation ceased to flow up to the medulla, and the reflex symptoms (*i.e.* tetanus), naturally subsided. Volkman has pointed out that irritation of a nervous centre, through a centripetal nerve, causes contractions, which last after the withdrawal of the stimulus. 2nd. A similar case rests upon the evidence of Baron Larrey. Tetanus followed an injury to the supraorbital nerve, and was directly cured by division of the nerve. This case is mentioned in most works on surgery. The same remarks apply to it as to Dr. Murray's case. 3rd. Bryant reports that Mr. Key, in 1845, amputated a leg for tetanus, which had followed on an unreduced dislocation of the astragalus. The symptoms disappeared at once after the operation. On dissection, the posterior tibial nerve was found to have been put violently on the stretch by the projecting astragalus. In this case there is no wound; septic influence is excluded; a peripheral nerve, the posterior tibial again, is kept violently on the stretch; irritation is propagated to the medulla; the train of reflex phenomena, known as tetanus, ensues; the source of irritation is cut off by the knife; the symptoms of reflex irritation cease immediately. Nothing could be plainer. I take these three cases as proving that peripheral nerve-irritation is a cause of the disease—the first step in this inquiry.

There are very many cases mentioned in surgical works, which go to strengthen the above proposition. Erichsen gives five or six cases, in which post-mortem examination revealed laceration, puncture, or other obvious injury of the nerve of the part. He seems to consider peripheral nerve-irritation as the starting-point of the disease in traumatic cases. Many other eminent surgeons agree in this. No one, as far as I know, has ever brought forward proof that any other cause exists. On the principle that like effects from like causes arise, this cause may safely be extended to all traumatic cases. Indeed, peripheral nerve-irritation is an essential feature of traumatism, and is included in the definition. Proposition the first being proved above, the following may be taken as a

corollary. Traumatic tetanus arises from peripheral nerve-irritation.

Let us see now what bearing this has upon so-called idiopathic tetanus. First, I must recapitulate that there is not an iota of direct proof that tetanus is ever idiopathic, whatever the word may mean. The term is here used simply in a negative sense. It is the negation of injury. If we accept peripheral nerve-irritation as the source of one form of tetanus, we must exclude it from the other, to justify the classification by negation. The question then arises—Is, or is not, peripheral nerve-irritation present in idiopathic tetanus? This question is embraced in the second proposition: viz. peripheral nerve-irritation is always present, which I will now endeavour to prove.

The great bulk of idiopathic cases are, in point of fact, either puerperal, menstrual, or the result of worms. They are put down as such in hospital records; this is well known, and easily proved. Indeed, there are authors—as MM. Laurent and Lombard—who go so far as to say that tetanus, even when traumatic, is almost always caused by worms. So common is this association of worms with tetanus at Madras, that *santonin* is a stock remedy in idiopathic tetanus. Again, puerperal and menstrual cases are exceedingly common. Looking over the records of the hospital, which I am now in charge of—the Women and Children's Hospital, Madras—I find that all the idiopathic cases which have been recorded in detail, including uterine and vaginal diseases, are either puerperal or menstrual, or the result of worms. Dr. King, formerly in charge of the Triplicane Hospital, Madras, found that out of 224 cases of tetanus treated in that hospital, 111 were males, 113 females. He makes the remark: "These numbers are nearly equal, showing that menstrual and puerperal predisposing causes in the one sex, about compensate the greater liability of the other to mechanical injury." In these acknowledged forms of idiopathic tetanus, peripheral nerve-irritation is undeniably at work. I have examined the gut (post-mortem) in cases the result of worms, and have found chronic inflammation, ulceration, worm-burrowing, and other signs of considerable local irritation. I have examined the utero-

vaginal tract in puerperal and menstrual cases, and have found congestion, erosion, ulceration, clots, and other signs of local irritation. To deny peripheral nerve-irritation in these cases, would be to deny that the parts concerned are supplied with nerves. It follows that in a large class, the greater part of idiopathic cases, peripheral nerve-irritation is at work. Now, if peripheral nerve-irritation at the surface causes tetanus, as has been proved to be the case, it is highly probable that it acts in the same way beneath the surface; that is, in the interior of the body.

There is but one step more in this inquiry, and that is to include all idiopathic cases. We must infer from the foregoing that any source of peripheral nerve-irritation within the body is capable of producing tetanus. Now, what are the possible sources of nerve-irritation within the body? Their name is legion. Parasites of all sorts, ulcers, concretions, tubercles, vomicae, morbid growths, &c., diseases of the lungs, liver, bladder, and other organs. In a word, any disease, anywhere in the body, is a source of nerve-irritation; and that case of idiopathic tetanus from which all such sources could be excluded must be absolutely free from disease. I cannot imagine that such a case has ever existed. Bryant says: "The late Mr. Wilson King, of Guy's, is stated by Poland to have been in the habit of remarking at the post-mortem table, whenever there was an examination of a case of death from tetanus, 'Gentlemen, we will now proceed to give you demonstration of a case of healthy anatomy; for there will be no visible morbid appearance otherwise than congestion of the organs in various degrees, which are owing to accidental circumstances.'" What did he mean by congestion of the organs "owing to accidental circumstances"? what organs, and what circumstances? Seeing that his remarks apply to traumatic cases, and perhaps to them chiefly, the organs referred to may be those of the nervous system, in which case the inference is clear. Anyhow, supposing his post-mortem examinations were more than usually searching, a vast number of sources of peripheral nerve-irritation in the organs, vessels, nerves, muscles, bones, and tissues generally, would, if present, escape detection. So that his remarks do

not in the least affect my second proposition that this cause, viz. peripheral nerve-irritation, is always present. This post-mortem question will be fully discussed under the heading of Proposition the 4th.

I now come to the third part of this inquiry: Does nerve-irritation give rise to the group of symptoms known as tetanus? If we were to reason from the known symptoms of the disease back to their hidden cause, it is precisely nerve-irritation that we would expect to find at the bottom of every case. The symptoms of tetanus are those of constant irritation of the excito-motor system, with great exaltation of the reflex function of the cord and medulla. It is characterised by muscular spasms, tonic and clonic, the latter brought on by the least noise or motion, the slightest stimulus to the peripheral nerves. The brain is clear and unaffected. There is often no fever, no constitutional disturbance of any kind. Physiologists know that if one of the spinal nerves of a headless turtle be irritated, contraction of the muscles follows on both sides of the body, both above and below the roots of the nerve operated upon. The irritation goes along the afferent nerve to the medulla, the centre of reflex irritability and seat of decussation of motor nerve fibres, from whence the impulse of motion is sent through the efferent nerves to any muscle in the body.

So wide-spread are the connections of the medulla that its afferent nerves are involved in every disease or injury within as well as without the body. Tetanus can be produced artificially in a headless frog, which has been rendered excitable by the injection of opium into the blood, by irritating the peripheral nerves (Kirke). Similarly with strychnine.

The pathology of tetanus would seem to be this:—  
1st. Continued irritation of the peripheral extremity of a nerve or *nervus nervorum*, through injury or disease of some sort.  
2nd. Continued irritation of the medulla, with tonic reflex spasm, *risus sardonicus*, and *trismus*.  
3rd. The unbroken cycle of continued irritation leads to morbid molecular changes, with violent disturbance of the reflex function and paroxysmal tetanus, on the least excitation, as a current of air.

Clinical facts support the idea that the medulla is early

affected. Nothing is more remarkable in tetanus than that it always begins with trismus, risus sardonicus, and stiffening of the sterno-mastoid muscles. These symptoms clearly denote irritation of the medulla, inasmuch as these muscles are supplied by nerves, the inferior maxillary division of the 5th, portio dura of the 7th, and spinal accessory, all of which arise from the medulla. Why these parts should be the first affected, is another question. It may be because they are nearest to the medulla, which thus expends its purposeless reflex force by the shortest route. Thus, for instance, if a drop of boiling water falls upon the hand, it is involuntarily withdrawn by a well-meant reflex act quicker than thought. But if the hand be well scalded, there is no meaning in the continued peripheral nerve-irritation which results, and which is a common cause of tetanus. Here we may suppose that the reflex irritation is transferred by the medulla to the nearest muscles by the shortest route, to expend the agony as it were, for nature perceives that to move the hand would now but increase the pain and reflex irritation. To avoid drawing out further the pathology of tetanus, which would be a digression in this place, I will conclude this branch of the subject by saying that Proposition the 3rd has been demonstrated experimentally. Peripheral nerve-irritation produces eventually the group of symptoms known as tetanus.

Fourth.—Does peripheral nerve-irritation, taken with the disturbance of the excito-motor function which it causes, account for the actual facts of morbid anatomy as found in post-mortem examination of patients dying of tetanus? Reflex irritation is a dynamical phenomenon. In its most distorted form we need not necessarily expect to find its track, post-mortem, as an objective morbid state. There is a strong antecedent probability that in some cases—acute cases of some duration—molecular changes do take place; but then such changes may exist with an undisturbed appearance of parts even with the aid of the microscope. Now there are many cases of tetanus on record in which no morbid change of any kind could be detected in the cord post-mortem. Bryant, Billroth, Nothnagel, and others—men who have the reputation of being careful observers—testify to this. Billroth says, “My

investigations of the spinal cord and nerves in tetanus have, as yet, given negative results only." This is not surprising, considering the nature of the disease. A great number of observers of equal weight, however, state that they have found marked changes, some in the cord, others in the medulla, others again in the floor of the 4th ventricle; and in traumatic cases inflammatory changes have been found in the peripheral nerves implicated. Under the microscope, parts apparently sound have been shown to have undergone change. At a late meeting of the Pathological Society, London, April 29, 1879, Dr. Ross showed microscopic sections of the cord after tetanus. He found dilatation and plugging of vessels, migration of leucocytes and of a granular material round the vessels, chiefly in the grey substance of the cord and in the grey nuclei in the floor of the 4th ventricle. Besides these inflammatory changes, he found the cells, especially the median group of the anterior horn of the columns, injured. Lockhart-Clarke, Erichsen, Allbutt, Gowers, Coats, Dickinson, and Benedikt have described similar changes. Aitken speaks of increase of specific weight; Rokitsansky and others of a development of fresh connective tissue. In two so-called idiopathic cases, one puerperal, the other the result of worms, which I examined lately, the following changes were found:—bright-red vascular patches on the dura mater over the medulla, colour persistent after repeated washings; vessels of the pia mater much engorged; arachnoid opaque and of a brownish tint, and excess of serous fluid. These changes are not in any way inconsistent with the proposition stated above; for although their absence does not militate against it, their presence is certainly more in accordance with what physiology would lead us to expect. Exalted function is always accompanied by hyperæmia. Increase of work necessitates increase of blood supply, and the line of demarcation between healthy plethora and morbid engorgement is often a very fine one and easily overstepped. Witness the condition of the breast at full time, itself a reflex phenomenon dependent on the condition of the uterus. We would expect to find post-mortem evidence in some cases of congestion, in others of inflammation and molecular disintegration; but we could not, from the nature of the

disease, expect to find such changes always, and this is exactly what happens. The length of time the disease has existed, and its intensity, should, moreover, influence the result. A mild case ending fatally in a very debilitated subject would not probably yield such marked changes as a severe case in a robust subject. The negro, mentioned by Dr. Robertson, who died in a quarter of an hour from spasm of the glottis (?), would not present any change at all. According to the view of the subject herein argued, in the first stage of the disease, that of continued nerve-irritation reacting upon the medulla and originating tonic spasms, up to a certain point there is no morbid change, and consequently there would be no evidence of disease. At this period division of the afferent nerve, by cutting off the source of irritation, arrests the symptoms. Afterwards, continued irritation exciting the reflex function to an inordinate degree, causes hyperæmia with molecular changes, and frequently inflammation. Here there may or may not be visible change, but there will be hidden molecular changes. Pathological facts fit in exactly with these views. Sometimes no changes are found, sometimes microscopical changes, and sometimes evidences of inflammation of the medulla and upper part of the cord. Proposition the 4th—peripheral nerve-irritation explains the facts of the morbid anatomy of the disease.

Here I must digress to say that I once thought that the disease itself was a species of neuritis ascendens; but the absence of post-mortem evidence of inflammation in many fatal traumatic cases, both in the peripheral nerves implicated and in the cord, makes this view untenable. Professor Nothnagel of Jena, who has specially studied this view of the question (*vide* New Sydenham Society's Second Series, 1877), has come to the conclusion that it cannot be proved either by clinical or pathological evidence, and he is forced to the conclusion that tetanus is due to an increase of nerve-irritability, possibly resulting from molecular changes beyond demonstration.

There remains the fifth proposition, that the peripheral nerve-irritation view of tetanus is borne out by its treatment. I have already brought forward three cases, in two of which, section of the nerve, and in one, amputation, cured the disease,

as a proof of its origin in nerve-irritation. These three cases are of equal value in proving any present proposition. Later on I stated that this treatment, by the knife, is only efficient up to a certain point; that when molecular changes take place in the medulla it is useless. Division of the nerve in the second stage may help, but does not cure the disease. Excision of the offending part does not affect it. Amputation of the limb is of no avail. All these have been tried and have failed signally. Something more is wanted—some powerful wrench to the nervous system that will alter the morbid molecular arrangement of the parts, or act as a revulsive or counter-irritant. Stretching a large nerve trunk and making traction on the cord, for instance, may prove of use. Well, this has been tried. The sciatic nerve has been stretched, and with marked success. I have seen it abolish clonic spasm for hours, in some acute traumatic cases, arising from injury to the limb operated upon. It may be said that this also acts by stopping the current of peripheral nerve-irritation. I think not. I think its action is upon the molecules of the spinal marrow or as a revulsive for this reason. The conductivity of the nerve itself is apparently unchanged. Sensation, nutrition, and motion, remain unimpaired in the limb operated on. Again, Prof. Annandale of Edinburgh lately stretched the spinal accessory nerve for spasmodic wry neck, without effect; whereas a cure immediately followed subsequent division of the nerve. In some cases of leprosy, and indeed in other special cases, the nutrition of the hand is improved by stretching the ulnar nerve. In 1878, Baum of Danzig cured a mimic spasm by stretching the facial nerve. Clearly then the nerve still conducts, but the mechanical violence gives rise to mechanical changes in the nerve centres or something of the kind, whereby reflex excitement is for a time subdued (?). If this view of the action of nerve-stretching is correct, it should act more or less well in all cases, whether puerperal or the result of worms, or what not, according as the afferent nerve implicated entered low down or high up the cord. I have made trial of this crucial test in a so-called idiopathic case (puerperal); but the case was hardly acute enough for my purpose. There was complete trismus, compelling rectal



alimentation ; but medicines were at times given by the mouth and swallowed. I saw no spasms of opisthotonos, but the attendants, apothecary and nurse, said that they had. I stretched the right sciatic nerve with a tension of 10 lbs.; there were certainly no spasms after the operation, and the patient ultimately recovered under the influence of chloral. The case goes to prove my proposition, but not so far as if it had been more acute. I will not venture to infer anything, however, from the result of this single case, the more especially as, since writing then, I have had a case which seems to contradict it. A girl aged eight years was seized with acute tetanus ten days after having the cartilage of her ear pierced for an ornament. I stretched the left sciatic up to 12 lbs. on the day of admission, and, with chloral, she improved very much ; but next day the clonic spasms were worse than ever. Seeing this, I opened the wound and stretched the sciatic again, but without the least effect. The girl died that night. Here failure may have been due to the fact that the sciatic nerve is a long way off from the cervical plexus, whose branches were irritated, but this is a separate question and an obscure one. It would have been better in this case to have stretched the cervical plexus. As regards treatment, the fact of greatest significance perhaps is, that whenever we look, we find men of all shades of opinion treating the disease as if it arose from peripheral-nerve irritation and had its climax in distortion of the reflex function of the medulla. Billroth himself originated nerve-stretching in traumatic cases. Surgeons almost invariably amputate fingers and toes when tetanus follows on their injury. And for constitutional treatment, the drugs used are just those which are known to diminish the reflex irritability of the cord, such as chloral, atropia, calabar bean, bromide of potassium, chloroform, alcohol, opium, and the like. And moreover, such drugs have justified their use in this disease. There is hardly one of them which has not been credited with a number of well-authenticated cures. They have each their special advocates ; but no one doubts that all in turn have from time to time successfully combated the disease. The ice-bag to the spine, leeches, blisters, and plasters, to the same, all testify to the universal opinion, acted

upon, if not avowed, that tetanus is a disturbance-disease of the reflex function of the cord. I need hardly dwell further on this point, the 5th proposition, It (the peripheral nerve-irritation theory) guides the treatment of the disease and is proved by its success.

I will now consider the force of these five propositions taken together, as evidence that peripheral nerve-irritation is the cause of tetanus.

If a number of murders took place in different parts of England, and all were of the same kind—by strangulation, for instance—and one man was always found present, no matter when the murder took place, strong suspicions would rest upon him, even though there was no direct proof of his guilt. But if subsequently this man was proved to be a Thugge, a man who had been convicted of at least three murders, and that by strangulation, a jury would surely find that he had been guilty of all. I am imagining a very strong case, one in which there is no evidence implicating any one else. Such is the nature of the evidence against nerve-irritation in tetanus. The patients all die in the same way. Nerve-irritation is always present. Nerve-irritation has been convicted in at least three similar cases. The patients die in just the way nerve-irritation would kill them. The post-mortem appearances are just such as it would leave behind it. There is no evidence to show any other cause of death. In this simile I lose the value of the 5th proof, or that by treatment, which tells very strongly; but I think that the evidence is already strong enough.

## HEREDITY AND CRIME IN EPILEPTIC CRIMINALS.

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I PURPOSE in the following tables to put together certain details relative to the hereditary history of those prisoners admitted into the Wakefield Gaol during the past three or four years, who were epileptics. The question is one of such interest in connection with the probable existence of a distinct criminal neurosis, that little apology, I think, is needed in bringing it to the attention of those specially interested in the study of mental disease. That there is some connection between crime and insanity few, doubtless, who have had any experience of either a prison or asylum would be prepared to deny, even if they were disinclined to go to the length of admitting that in many cases the two are distinctly correlated.

In making any inquiry into the history of prisoners there are three main difficulties to contend with. First: the well-known mendacity of the criminal classes generally. Secondly: the meagre and imperfect knowledge they usually possess of their relatives; and, Thirdly: the common occurrence of illegitimacy. In such an inquiry as the present the first difficulty is naturally one of the greatest moment.

It would occupy a needless amount of space to detail the precautions that have been taken in making these investigations, but it seemed right to put this great difficulty prominently forward, with the assurance that, so far as was practicable, every possible care has been taken to test the truth of the statements contained in the following pages.

Members of the class from which our criminals are mostly drawn commonly get separated from the parent stock very

early in life, and, in the subsequent struggle for existence, having to fight their way apart, rapidly lose all sight, and frequently all recollection, of even their immediate ancestors. In at least 5 per cent. of the cases examined the prisoners had no knowledge whatever of any member of their families. This difficulty applies especially to the Irish, who leave their country when comparatively young, and frequently never see or hear of any of their relatives again. As the Irish form about one-sixth of the whole number of prisoners admitted, it will be seen that this drawback is one of some moment. Perhaps the only compensating advantage is that the average results will be understated rather than exaggerated. The third difficulty is one that undoubtedly comes into frequent play, but is of such minor importance in comparison with the other two that it may be dismissed with the remark that inquiry has been made, and care taken to make such allowances for it as were necessary.

Out of a total of 119 cases examined, 89 were males and 30 females. It is usually taught that among the general population epilepsy attacks the two sexes in about equal proportion.<sup>1</sup> There is reason to think that with criminals the same rule holds good. During the year 1877 there were committed a total of 9672 males and 2832 females, or 1 female to every 3·3 males, a proportion, it will be seen, very similar to that between the two sexes among the epileptics.

The cases have been divided broadly into two classes—idiopathic and traumatic. This division is somewhat arbitrary; but the two classes exhibit a marked difference, both in hereditary and personal history. Any attempt to ascertain the exciting cause for epilepsy is generally a matter of difficulty even in intelligent patients; under the term traumatic, therefore, are classed only those cases in which the attack commenced for the first time within a month at least of some sudden psychical or physical accident. Under the head of idiopathic are placed all those where the fits had existed from birth, or commenced later on in life without the intervention of such rapidly acting external agents. As idiopathic cases, therefore, I have included all those assumed

<sup>1</sup> Reynolds's 'System of Medicine,' vol. ii. p. 295.

to be due to long-continued habits of intemperance on the part of the patients themselves; and this the more readily because certain forms of alcoholism with resultant epilepsy are undoubtedly due to such hereditary predisposing causes as, under other circumstances, or in individuals differently constituted, would have directly produced the convulsive attacks. Dr. Savage has seen cases that convinced him that drink-craving has been the result of nervous disease in one or other parent.<sup>1</sup> This division applies only to males. The ascribed traumatic causes among females were so few and slight that, for the sake of simplicity, they have all been counted as idiopathic.

Among the traumatic cases, 26 in number, 15 were said to be due to injury to the head, 5 to fright, 4 to sunstroke, and 2 to injuries of an undefined nature. These two last occurred early in life. In neither was there any distinct history of head-injury, but, in both, the fits followed immediately after the accident.

The ages of the epileptic prisoners are given in Table I., and in Table II. the ages of the 3341 prisoners of all classes committed during the quarter ending March 31st, 1878.

TABLE I.

TABLE showing the AGES of 119 EPILEPTIC PRISONERS.

AGES.	IDIOPATHIC.						TRAUMATIC.		Net Total.	
	Males.		Females.		Total.		Males.			
	No.	Percent.	No.	Percent.	No.	Percent.	No.	Percent.	No.	Percent.
16 and under 21	4	6·4	1	3·3	5	5·3	2	7·7	7	5·8
21    „    „    30	13	20·6	10	33·4	23	24·8	6	23·1	29	24·4
30    „    „    40	26	41·3	11	36·7	37	39·8	6	23·1	43	36·2
40    „    „    50	11	17·4	4	13·3	15	16·1	8	30·7	23	19·3
50    „    „    60	5	7·9	4	13·3	9	9·7	4	15·4	13	10·9
60 and upwards	4	6·4	..	..	4	4·3	..	..	4	3·4
Total. .	63	100·0	30	100·0	93	100·0	26	100·0	119	100·0

<sup>1</sup> 'Guy's Hospital Reports,' 1877, p. 68.

TABLE II.

TABLE showing the AGES of 3341 PRISONERS committed during the QUARTER ending MARCH 31st, 1878.

AGE.	Males.		Females.		Total.	
	No.	Per cent.	No.	Per cent.	No.	Per cent.
Under 12 years of age . . .	6	0·2	1	0·1	7	0·2
12 and under 16 . . . .	41	1·5	18	2·7	59	1·8
16 „ „ 21 . . . .	301	11·2	110	16·5	411	12·3
21 „ „ 30 . . . .	974	36·4	204	30·7	1178	35·3
30 „ „ 40 . . . .	674	15·2	175	26·3	849	25·4
40 „ „ 50 . . . .	361	13·5	115	17·4	476	14·2
50 „ „ 60 . . . .	221	8·3	35	5·3	256	7·7
60 and upwards . . . .	98	3·7	7	1·0	105	3·1

A comparison of these two tables will show that the decennial period between 21 and 30 includes the larger number of ordinary prisoners, while that between 30 and 40 takes the greatest percentage of epileptics. The average age for epileptic males is 37·0; and for females 34·8. This greater average age for epileptic prisoners may be partly due, as will be seen further on, to their more prolonged and persistent criminal career, as indicated by the greater number of recidivists among them. The large percentage in the decennial period between 40 and 50 among the traumatic males is probably accidental, the number of cases being too small to furnish any decisive information.

Table III. shows the ages at which the fits commenced. Table IV. is taken, for the purposes of comparison, from Dr. Bennett's Clinical Lectures, and Dr. Reynolds's article in 'The System of Medicine.'<sup>1</sup>

<sup>1</sup> Vol. ii. p. 296.

TABLE III.

TABLE showing the AGES at which the FITS commenced in CRIMINAL EPILEPTICS.

AGE.	IDIOPATHIC.						TRAUMATIC.	
	Males.		Females.		Total.		Males.	
	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
Under 10 years . .	20	31·7	9	30·0	29	31·2	2	7·7
From 11 to 20 . .	18	28·5	5	16·7	23	24·7	8	30·8
„ 21 „ 30 . .	14	22·2	8	26·6	22	23·6	7	26·9
„ 31 „ 40 . .	8	12·7	3	10·0	11	11·8	4	15·4
„ 41 „ 50 . .	2	3·1	5	16·7	7	7·5	4	15·4
Over 50 . . . .	1	1·6	..	..	1	1·1	1	3·8

TABLE IV.

TABLE showing the AGES at which the FITS commenced in Ordinary EPILEPTICS.

DR. BENNETT'S CASES.				DR. REYNOLDS'S CASES.			
AGE.	Males.	Females.	Total.	AGE.	Males.	Females.	Total.
From 1 to 10 .	9	14	23	Under 10 . .	10	9	19
„ 10 „ 20 .	11	23	34	From 10 to 20.	66	40	106
„ 20 „ 30 .	14	9	23	„ 20 „ 45.	25	20	45
„ 30 „ 40 .	10	6	16	Over 45 . . .	1	1	2
„ 40 „ 50 .	1	..	1				
„ 50 „ 60 .	2	1	3				
Totals . .	47	53	100	Totals . .	102	70	172

From the latter table it appears that in ordinary epilepsy the majority of cases commence between the tenth and twen-

tieth years of life, the numbers in the decennial period on either side of this being about equal. Trousseau also gives the first place to the second ten years of life, but states that a larger number of cases occur in the period before than in the period after.

The particulars in the column for traumatic cases in Table III. are of little interest. Being directly due to outside causes, the fits, as one would naturally expect, occur for the first time during those years of life when the body is most exposed to external injury. Those under ten years of age were the two boys already mentioned, and having a doubtful traumatic origin. Among the idiopathic cases the most noticeable point is the larger percentage commencing under ten years of age, which, in my own cases at all events, practically means that the fits had existed from childhood. M. Lancereaux found with regard to fits as the result of hereditary alcoholism that, in children who had survived the dangers of convulsions in infancy, epilepsy not unfrequently broke out at the period of puberty.<sup>1</sup> Three or four of my own cases go in support of this statement, an interval of several years elapsing between the cessation of the convulsions and the development of epilepsy proper.

In suggesting any explanation of this earlier age for the commencement of epilepsy among criminals, it should be borne in mind that the disease occurring early in life must be a great hindrance to the sufferers being brought up to any useful trade or mechanical employment, or to their competing with others who are not so afflicted; so that with the ordinary means of subsistence lessened, frequently with a tendency to weak-mindedness, and with no relatives capable of supporting them, it is little to be wondered at that they should gradually gravitate into a state of vagrancy and beggary. In a mining district like the West Riding this probably holds good to a greater extent than it would in an agricultural county. Many of these criminal epileptics are well-known characters, and spend the greater part of their life fluctuating between the workhouse and the prison.

Case No. 4. Male, æt. 33. Has had fits since he was 14 years

<sup>1</sup> 'Gazette des Hôpitaux,' April 26, p. 377.



old. They are of such frequency and severity that he has had to spend the greater part of his subsequent life in the work-house, as no one would keep him employed for any length of time. He bites his tongue severely, and his face and head are profusely scarred from injuries he has received. They always commence with a prickling sensation in the right hand. Whenever he thinks a fit is coming on, he gets drunk, if possible, with the view of warding it off, but he does not think it has the desired effect. Father died suddenly thirteen years ago; was not a heavy drinker. Mother is living; neither drinks nor has fits. Brother was a heavy drinker. Youngest sister had a running from the ears and was "troubled in her head"—probably imbecile. Eldest sister had eleven children, all of whom died when three or four days old, mostly in convulsions. He himself is a drunkard, and has been committed to prison four times as a disorderly pauper, twice for larceny and twice for assault.

Case No. 90. Male, æt. 47. Had convulsions in childhood, which were said to be due to "water on the head." He has since been "affected in his nerves throughout his body." He gets his living now by singing hymns about the street. Before that he was kept at home, never having been taught any trade. About nine or ten years ago he had a stroke and was paralysed "all over his body," so that he was unable to hold a pen or do anything for himself. Prisoner is of weak intellect and has right hemiplegia. He has no settled residence and remembers little or nothing about his family. He has not seen any of them now for many years; but has heard that his father was a moderate man and died of old age, and that his mother died when he was a child. He is committed to this prison as a disorderly pauper. Has been in other prisons, but does not remember how many times.

It will be seen, on referring to Table XI., that the convictions under the heads of rogues and vagabonds, vagrancy and disorderly paupers are more numerous among epileptics than non-epileptics. Again, it will be shown in the next paragraph that a very frequent probable predisposing cause of epilepsy among criminals is drunkenness on the part of the parents. It is now a well-established fact that the children of drunkards are

especially liable to convulsions in infancy. M. Martin has lately investigated this question at the Salpêtrière. In his first group of 60 epileptic patients, one-fifth of the children had convulsions, and more than one-half died early. His "83 families, in which one or more members suffered from epilepsy of alcoholic origin, had 410 children; and of this number, 108 (more than one-fourth) have had convulsions; and in 1874, 169 were dead, 241 lived, but 83 (more than a third of the survivors) were epileptics."<sup>1</sup> It has been remarked by Dr. Reynolds that "where there is a marked hereditary taint as a predisposing cause of epilepsy, the disease is found to develop itself somewhat earlier than under other circumstances."<sup>2</sup> The difference for ordinary epileptics is represented in the following table:—

Commencing under æt. 15 . . .	83·33 hereditary	46·15 non-hereditary.
„ above æt. 15 . . .	16·66 „	53·82 „

The same rule is found to hold good with respect to criminals, but the difference is somewhat less marked. The distinction with regard to sex, on the other hand, is rather in accordance with the results arrived at by Messrs. Leech and Fox; hereditary epilepsy in criminals shows itself on an average four years earlier among men than among women.

Unfortunately, one of the most prominent features in the hereditary history of these prisoners is one concerning which there is the greatest difficulty in arriving at the truth with any degree of exactitude. The amount of intemperance thought sufficient to constitute a "drunkard" is found to vary very materially in the minds of different individuals. The "heavy drinker" of one son, is the father who "only gets drunk every Saturday night" of another, a practice thought to be so little out of the common, that the man is called "sober." As this seemed to be one of the most interesting points in connection with the subject, precautions have been taken to include under the head of "drunkards" none but those in whom the habit was marked and confirmed. Two classes of people have been specially noted, namely those described as "scarcely ever sober," and those subject to paroxysmal attacks

<sup>1</sup> 'BRAIN,' July 1879, p. 293.

<sup>2</sup> *Op. cit.* p. 296.

of an uncontrollable character, with intervals of sobriety or even total abstinence; cases, in short, where the habit has become distinctly morbid. In ordinary phraseology, nearly all those marked as doubtful would come under the head of "drunkards." The difficulty in estimating the amount of drunkenness in individuals is so great, that some authors think it right to reject such evidence altogether. By so doing they appear to me to exclude one of the most valuable points in the inquiry. As it is more conceivable that patients should conceal drunken habits in other members of their family, rather than exaggerate or invent them, the tables will have at least the negative merit of understating the probable truth. As a matter of fact, in most cases the father was found to be the great drunkard of the family. A history of drink on the part of the mother was but rarely met with. This is probably simply an indication of the greater sobriety of the female half of the population, though the greater reticence of the prisoners with regard to their mothers' failings may have something to do with it.

TABLE V.  
HABITS of the FATHERS of EPILEPTIC CRIMINALS.

	IDIOPATHIC.						TRAUMATIC.		Net Total.	
	Males.		Females.		Total.		Males.			
	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
Fathers drunkards	35	55·6	11	36·7	46	49·5	7	26·9	53	44·6
Fathers sober . .	21	33·3	9	30·0	30	32·3	11	42·3	41	34·4
Unascertained or doubtful }	7	11·1	10	33·3	17	18·2	8	30·8	25	21·0
Total . .	63	100·0	30	100·0	93	100·0	26	100·0	119	100·0

In addition to the details given in the above table, 15 idiopathic males—23·8 per cent.—gave a history of drunkenness in 24 other members of their family, distributed thus:—Grandfather, 1; mothers, 3; uncle, 1; aunt, 1; brothers, 18.

Three idiopathic females—10 per cent.—gave a history of drink in three other members of their family; in one case a mother, and in the other two brothers. One traumatic male said his mother had been accustomed to drink heavily, and had committed suicide while under its influence. One of the females' fathers also committed suicide.

The difference between the idiopathic cases and the traumatic is most marked; the former giving a total of 49·5 per cent. of fathers as drunkards, the latter only 26·9 per cent. One other point worthy of note in this table is that the percentage of drunken fathers among the males is greatly in excess of that among the females. Probably many of the doubtful cases among the latter should by right be included; but, making allowance for this and for the possible greater amount of drunkenness among the mothers, an hereditary history of alcoholism is certainly more frequent among epileptic men than women. This may be partly compensated for by the increased number of epileptics among the ancestors of the females. (See Table X.)

It is difficult to estimate the importance of this table without having some notion of the frequency of drunkenness in the parents of criminals who are non-epileptic. The only figures I have, selected with the same stringency, are those of 30 females, taken consecutively for this purpose; they give the following results:—

Fathers drunkards . . .	7 = 23·3 per cent.
Fathers sober . . . . .	15 = 50·0 „
Unascertained, or doubtful .	8 = 26·7 „
Mothers drunkards . . .	2 = 6·6 „
Mothers sober . . . . .	23 = 76·7 „
Unascertained, or doubtful .	5 = 16·7 „

Among these women, then, with a less number of doubtful cases, there is a decrease of 13·4 per cent. of drunken fathers, and an increase of 20 per cent. of those who were said to be sober. A comparison of the two columns, idiopathic and traumatic in Table V., shows among males a still greater difference in the percentage of drunken fathers.

My colleague, the Rev. R. Bullock, Chaplain to the Prison, has kindly compiled for me a table of the presumed habits of

the fathers of 225 male prisoners committed for 6 months and over. This table gives:—

Fathers drunkards . . .	97 = 43·5 per cent.
Fathers sober . . . . .	126 = 56·5 „

The comparison perhaps is hardly just, as men committed for this length of time belong, as a rule, to a different class from those committed for short periods; but the difference is 12·1 per cent. in favour of the non-epileptics, a difference, it will be observed, remarkably similar to that found to exist between epileptic and non-epileptic criminal females. If equal stringency had been exercised in apportioning the habits of the fathers in these cases, the difference would in all probability be found to have been still greater on the side of the men.

Mr. Bullock has also furnished me with the statistics of all the boys committed to reformatories during the years 1872 to 1876 inclusive. The total number of boys was 730; those said to have had drunken fathers being 194, and drunken mothers 29. This will give a percentage of 26·5 for the former, and 3·9 for the latter. The percentage of drunkards among the mothers is, it will be observed, only slightly lower than it was shown to be among the idiopathic epileptics. I was prepared to find a considerably greater difference in the number of drunken ancestors between epileptic and non-epileptic criminals than was really proved to exist. There is reason, however, to believe that a hereditary history of alcoholism is more common, not only in epileptic but in ordinary prisoners than it is in non-criminal epileptics. Of 95 ordinary epileptics examined by M. A. Voisin, 12 had scrofulous or true tuberculous antecedents, and 12 had antecedents who died from alcoholic excesses, or were subject during their honeymoon to excessive abuse of alcohol.<sup>1</sup> This puts the proportion of drunken parents at only 12·6 per cent. Reasoning from the above facts, it is difficult to avoid the conclusion that both crime and epilepsy may owe their origin to alcoholism in the parents as a predisposing cause; in other words, that criminal instincts and epileptic convulsions are not unfrequently distinctly correlated. Nor do I see any reason to confine the term “criminal

<sup>1</sup> ‘London Medical Record,’ January 15th, 1878, p. 9.

instincts" in these cases to a tendency to alcoholic excess. Among the idiopathic males, there are 26 cases where the habits of the fathers were ascertained without there being any epilepsy or mental disease in the family. The average number of convictions for those with drunken fathers, as compared with those whose fathers were sober, is as 3·5 to 2·3, showing an increased amount of crime associated with hereditary alcoholism.

The statement that epilepsy in children is due to conception occurring while the parents are under the influence of drink, is one that must be naturally difficult of proof. I failed to get any evidence of the fact, nor do the conditions at all seem to require it. It is not suggested that the hereditary transmission of epilepsy is due to conception taking place during a paroxysm; the hereditary transmission of alcoholism then, which in some of its forms is surely an allied neurosis, would be guided by the same laws and occur under like conditions.

I pass on now to consider other forms of hereditary neurosis, the most important of which is naturally epilepsy itself.

*Direct* heredity in the case of criminals can seldom be traced beyond the father or mother. No general value therefore is to be attached to the few particulars given of grandparents.

*Collateral* heredity is confined mostly to brothers, sisters, uncles, aunts, and first cousins.

The idiopathic epileptic males give, including both direct and collateral relatives, a family history of fits in 29 cases, or 46 per cent. This includes one case in which the prisoner's children only were affected. The females give a like history in 21 cases, or 70 per cent., including two in which children only were affected; and the traumatic males in 4 cases, or 15·3 per cent. The total number of idiopathic cases giving a history of fits in one or more members of their family is then 50, or 53·7 per cent.; and the net total for all cases 54, or 45·3 per cent. Counting only direct relatives, we get a percentage of 25·8, 32·4, and 12·5 in the three classes respectively. The details are given in Table VI.

TABLE VI.

DETAILS of HEREDITARY HISTORY of FITS (omitting CHILDREN).

## IDIOPATHIC CASES.—MALES.

Relative.	No.	Per cent.	Degree of Relationship when ascertained.	Remarks.
Grandfather . . . . .	1	1·6	Mother's father.	
Grandmother . . . . .	1	1·6	Father's mother.	
Fathers . . . . .	4	6·4		
Mothers . . . . .	10	16·2	.. ..	1 "died of fits."
Brothers . . . . .	14	22·6	.. ..	8 "
Sisters . . . . .	13	20·9	.. ..	5 "
Uncles . . . . .	4	6·4	{ 3 were Mother's brothers. }	1 "
Aunt . . . . .	1	1·6	Mother's sister.	
Cousins . . . . .	3	4·9	{ 1 on Mother's side. 1 Father's brother's daughter. }	
Nephews and Nieces .	11	17·8	Sister's children.	{ All died in Infancy. }
Total No. of Relatives with Fits . . . . .	62	100·0		

## FEMALES.

Relative.	No.	Per cent.	Degree of Relationship when ascertained.	Remarks.
Fathers . . . . .	4	11·7	.. ..	1 "died of fits."
Mothers . . . . .	7	20·7	.. ..	1 " "
Brothers . . . . .	7	20·7	.. ..	2 " "
Sisters . . . . .	6	17·7	.. ..	1 " "
Uncles . . . . .	4	11·7	{ 2 were Mother's brothers. 1 was Father's brother. }	
Aunts . . . . .	4	11·7	{ 2 were Mother's sisters. }	
Cousin . . . . .	1	2·9	Female.	
Nephew . . . . .	1	2·9	Brother's child.	"Died of fits."
Total No. of Relatives with Fits . . . . .	34	100·0		

## TRAUMATIC CASES.

## MALES.

Relative.	No.	Per cent.	Degree of Relationship when ascertained.	Remarks.
Grandfather . . . .	1	12·5	Mother's father.	
Brothers . . . . .	4	50·0	.. ..	2 "died of fits."
Sister . . . . .	1	12·5		
Total No. of Relatives with Fits . . . . }	8	100·0		

## PROPORTION OF SEXES among RELATIVES AFFECTED.

IDIOPATHIC.		TRAUMATIC.
Males.	Females.	Males.
Per cent.	Per cent.	Per cent.
Males . . . 23 = 37·1	Males . . . 16 = 47·1	Males . . . 5 = 83·3
Females . . . 26 = 42·0	Females . . . 18 = 52·9	Females . . . 1 = 16·7
Unknown . . . 13 = 20·9		

Of the 29 cases among the idiopathic males in which epilepsy was present in the family, it existed in the father in 1; mother in 3; grandfather in 1; grandmother, mother, sister, brother, uncle, and child in 1; father and sister in 1; father and two sisters in 1; mother and sister in 1; mother and brother in 2; mother and five or six brothers and sisters in 1; mother, sister, and brother in 1; mother and aunt in 1; brother in 4; sister in 2; brother and cousin in 1; brother, uncle and two great-uncles in 1; sister and uncle in 1; uncle in 1; cousin in 2; ten or eleven nephews and nieces in 1; four children in 1.

Among the 21 idiopathic females, epilepsy existed in the father in 1; mother in 4; father, sister, cousin and child in 1; father, brother and child in 1; father and sister in 1; mother and child in 1; mother, brother and uncle in 1; mother and



brother in 1; brother in 1; brother, sister and child in 1; brother and aunt in 1; brother, sister, nephew and two aunts in 1; two sisters and uncle in 1; aunt in 1; uncle and six children in 1; two children in 1; three children in 1.

Among the 4 traumatic males, epilepsy existed in the grandfather, brother, and two children in 1; brother in 1; two brothers in 1; sister in 1.

With respect to numbers only, the 124 relatives were divided among the 54 epileptics as follows:—

#### IDIOPATHIC MALES.

1	had	11	relatives	affected.
2	"	5	"	"
1	"	4	"	"
2	"	3	"	"
8	"	2	"	"
15	"	1	relative	affected.

#### IDIOPATHIC FEMALES.

1	had	4	relatives	affected.
3	"	3	"	"
5	"	2	"	"
11	"	1	relative	affected.

#### TRAUMATIC MALES.

2	had	2	relatives	affected.
2	"	1	relative	affected.

An examination of the above tables, having regard to the idiopathic cases only, suggests the following conclusions: First; both among males and females epilepsy is more frequent in the mother than in the father: and secondly; the percentage for both parents is higher with the women than it is with the men. On comparing the above figures with those in Table V., it will be seen that this result is exactly the converse of what holds good with respect to drunkenness. There the male parent was found to be affected to a much greater extent than the female, and the percentage for both parents was higher among men than women. According to Trousseau, in ordinary cases hereditary predisposition is particularly apparent in persons descended from epileptic mothers.<sup>1</sup> The percentage of epileptics among brothers and sisters is slightly higher for

<sup>1</sup> Trousseau, Syd. Soc. Translation, 1879: Vol. iv. p. 357.

men than women, notwithstanding the fact that the number of direct hereditary epileptics is much greater among the latter. This seems to favour the suggestion previously made that certain forms of alcoholism in the parents are almost as efficient a predisposing cause for convulsions in the children as epilepsy itself; the number of drunken parents being so much greater among males than females. In connection with this point, and also as showing the great tendency of alcoholism to produce early convulsions in the offspring, it may be mentioned that the average age for the commencement of the fits, for those epileptics who have a direct hereditary history of drink, is less by  $4\frac{1}{2}$  years than for those whose parents are returned as sober.

Epilepsy in the father was usually found in connection with drunken habits. Out of 9 cases, where all the particulars were ascertained, epilepsy in the mother was associated with drunkenness in the father in 6, and the result was invariably intemperance on the part of the prisoner himself.

The large proportion of epileptics among the brothers and sisters in the traumatic cases would show that, apart from the exciting cause, there is often a marked hereditary predisposition. M. Lancereaux holds, with regard to alcoholic epilepsy, that the hereditary tendency of drunkenness manifests itself in the offspring in the form of excessive reflex excitability. He cites the case of a young boy who had severe epileptiform fits, lasting, with intermissions, for several hours, as the result of intestinal worms, the unusual severity of the attack being due to parental alcoholism.<sup>1</sup>

Case No. 3. Male, æt. 24. Was healthy until two years ago, when he got his head crushed by some machinery. Was removed to the County Infirmary, and five days afterwards he had his first fit. The fits have since occurred on an average about once a fortnight. They come on both day and night, and he has cut his face and bitten his lips several times. About two months ago he woke up one morning and found himself paralysed on the right side. His father was a heavy drinker. Two brothers died, æt. 7 and 18, both of whom had fits. Has an uncle who was in an asylum, and a cousin who is

<sup>1</sup> 'Gazette des Hôpitaux,' April 26.

there now. Acknowledges to having been a heavy drinker himself. Is in prison for the first time for larceny.

Case No. 17. Male, æt. 19. Twelve months ago he fell 7 feet on to his head, and was insensible for some time afterwards. Two days after the fall he had his first fit. They come on now at intervals of three or four weeks, and last but a few minutes. He bites his tongue freely, and has fallen down and cut his head more than once. Father is a heavy drinker. Mother living; does not have fits. One brother, aged 30, has had fits all his life. Prisoner himself is a heavy drinker. Has been committed to prison for damage, larceny, and drunkenness.

Case No. 18. Male, æt. 58. Was in the Indian Mutiny in 1858, and received a wound on the head; five or six years ago he fell and cut his head again in the same place. While the surgeon was examining this wound he had his first fit. The wound was followed by erysipelas. The fits have occurred since at very variable intervals, sometimes two or three a day, at others only one in four or five weeks. They last but a minute or so, and come on mostly in the night time. He bites his tongue and lips freely, and has cut his head severely. At the time of, or shortly after the fit, he is very violent, striking and biting any one near him. In consequence of these attacks of excitement he has been confined in an asylum on four different occasions. Father was lost at sea; was not a heavy drinker. Mother was a drunkard and drowned herself. She used to have fits, which, from prisoner's description, were probably attacks of *petit mal*. Has three brothers and one sister, the latter is subject to fits. His maternal grandmother was in an asylum. Is committed to prison for drunkenness. Has been in twice before, once for disorderly behaviour and once for assault.

Case No. 19. Male, æt. 45. When 19 years of age he had a fall from the rigging of a vessel; broke four ribs and his collar-bone, and injured his back and head. Was in his first fit when taken out of the water. Had a rapid succession of fits for four weeks, and was scarcely in his senses for three months afterwards. The fits have gradually lessened in frequency since the accident, and they now only occur once or twice in the month. He never bites his tongue, but has scars all over his

face and hands from injuries received in falling. Has had his face paralysed for some days after a fit. There is partial paralysis existing on the left side now. Mother dead 18 years from rheumatism. Father living; is a heavy drinker, and has been so for many years. His maternal grandfather had fits. A younger brother also had fits, and died from "disease of the brain and falling sickness." Sister has been in prison. Prisoner himself had two children who died in convulsions. He is a heavy drinker. Has been in this prison five times; once for larceny, once for hawking without a certificate, and three times for drunkenness. Has also been in Leeds Gaol for drunkenness.

Dr. Bennett in his analysis of 100 cases gives a family history of epilepsy in 26 per cent.; the father and mother being most frequently affected, and the brothers and sisters next in about equal proportion. It seems probable, therefore, that both hereditary and acquired epilepsy is far more common among criminals than it is among the general population. Cause and effect alternate in successive generations. As the parental taint of epilepsy may develop itself as criminal instincts in the offspring, so there is every reason to suppose epilepsy in the children may have its hereditary predisposition in some forms of habitual crime on the part of the parent. "The hereditary predisposition of an epileptic," says Trousseau, "may be traced merely to strange, nervous phenomena, perfectly different from epilepsy itself, whilst similar disorders may alone be manifested by his posterity, direct or indirect."<sup>1</sup>

The very large amount of hereditary neurosis that is frequently met with in these cases is somewhat remarkable.

Case No. 20. Female, æt. 22. Had convulsions in childhood, from which she recovered. Since her pregnancy has been subject to fits. Was under observation in the hospital for some time, and there is no question as to their epileptic character. Father was a drunkard. Mother living; neither has fits nor drinks heavily. Prisoner's maternal grandmother was insane. One brother had fits, and died in one when aged 22. One sister has had fits since childhood. Two sisters have

<sup>1</sup> Trousseau, New Syd. Soc. Translation. Vol. i. p. 87.

been in prison for drunkenness. Has two aunts now living who have fits. A cousin—mother's sister's child—is now in Hull Asylum. One of brother's children died in convulsions at 5 years of age. Prisoner herself is an intemperate woman, and has been in prison 13 times, including 3 for drunkenness. From her own statement most of the other offences were committed under the influence of drink.

The proportion of sexes among the epileptic relatives is shown in the latter part of Table VI. The 13 returned as unknown, in the first column, include 11 sisters' children in one case, all of whom died in infancy from convulsions. It will be observed that under the head of idiopathic, the number of females affected is slightly higher for both sexes. This also is the converse of what holds good with regard to drunkenness.

As I am mainly concerned with the hereditary causes of epilepsy among these criminals themselves, any neurosis in their children has only been incidentally alluded to. The following cases, however, may serve, in passing, to show the manner in which they, in their turn, are not uncommonly found to be affected. That the children of epileptic criminals are more liable to convulsions and other nervous affections than the children of ordinary epileptics, naturally follows from the increased amount of heredity that is found to exist among the epileptics themselves.

Case No. 10. Female, *æt.* 40. Has had fits for 6 years. No cause suggested, except fretting from prolonged imprisonment. They come on at very variable intervals, and nearly always in the night time. Never bites her tongue, but has cut her head severely in falling. Father dead 21 years; was not a heavy drinker. Neither of her parents, nor any other member of her family had fits. Prisoner herself is a drunkard. Has been in prison 14 times, including 7 for drunkenness, and two sentences of penal servitude of 5 and 7 years respectively. Her eldest daughter is a bad character, and has been in prison several times. Three other children died young from convulsions.

Case No. 4. Female, *æt.* 46. Has had fits 5 or 6 years. Does not remember either her father or mother. Has been in

prison 18 times, including 12 for drunkenness. Has only two children, and both are epileptic.

Case No. 108. Female, æt. 34. Has had fits since childhood. They have gradually lessened in severity, and are now only attacks of *petit mal*. She was early attacked with hemiplegia, and is still partially paralysed on the left side. Her uncle and father's brother were subject to fits; the latter is now in an asylum. Has had 8 children, 6 of whom died in convulsions when young. Prisoner was tried at the Assizes for attempting to murder her child, and acquitted on the ground of insanity. She had acute melancholia, with delusions, and on several occasions attempted to commit suicide.

Case No. 31. Male, æt. 52. Has had fits since he was 10 years old. They only come on about three or four times a year; he has cut his head in falling, but does not bite his tongue. Father was a heavy drinker in his younger days. Mother has been dead 30 years from paralysis. Had 3 sisters and 1 brother, but has not heard or seen anything of them since he was a child. Has been in prison 5 times; 4 for drunkenness and once for assault. Had 5 children; 4 died in childhood from fits, and the fifth, a son æt. 25, is now in an asylum. (See Table VII.)

Of the 16 cases among the idiopathic males in which insanity was present in the family, it involved the mother's cousin and mother's brother's child in 1; the mother's mother, and mother's sister in 1. Of the 9 cases among the females, it existed in the father's brother and mother's sister in 1; in the mother's mother and mother's sister's child in 1. Among the 3 traumatic males, a first and second cousin were affected in 1, and an uncle and cousin in 1. All the rest had each but a single member of the family reported as insane.

Having found the proportion of epileptic relatives to be so much greater with criminal than with ordinary epileptics, it was only to be expected that inquiry into their hereditary history with regard to insanity should give us a similar result. Dr. Bennett's investigations show only 5 per cent. of ordinary epileptics with insane relatives, as against 24 per cent. of my own cases. The figures in the above table are too few to furnish any satisfactory general conclusions, but it may be

TABLE VII.

TABLE showing the details of INSANITY in the families of EPILEPTIC CRIMINALS.

RELATIVES INSANE.	IDIOPATHIC.				TRAUMATIC.	
	Males.		Females.		Males.	
	No.	Degree of Relationship.	No.	Degree of Relationship.	No.	Degree of Relationship.
Grandfather	1	{Mother's father (committed suicide).}				
Grandmother	1	Mother's mother.	1	Mother's mother.		
Fathers .	2	.. ..	1			
Mother . .	1	{(Died insane and paralysed.)}				
Uncles . .	4	{2 were Father's brothers.}	3	{2 were Father's brothers. 1 Mother's brother.}	2	{1 was Mother's brother.}
Aunts . .	3	{2 were Mother's sisters.}	3	{2 were Mother's sisters.}		
Brother . .	1					
Sister . .	1	{(? Congenital im- becile.)}				
Child . .	1	Son.				
Cousins. .	3	{1 Mother's bro- ther's child. 1 Mother's cousin.}	3	{1 Mother's sister's child. 1 Father's cousin.}	3	(1 an imbecile.)
Total No. of Relatives Insane . }	18		11		5	
Proportion of Sexes among Insane Rela- tives . . }	Per cent. Males . . 9 = 50·0		Per cent. Males . . 4 = 36·4		Per cent. Males . . 2 = 40·0	
	Females . 6 = 33·4		Females . 4 = 36·4			
	Unknown . 3 = 16·6		Unknown 3 = 27·2		Unknown . 3 = 60 0	

remarked that, both with males and females, the uncle, aunt, and cousin are most frequently affected; that among the men the hereditary history of insanity is greater on their own side, while among the women it is equal for the two sexes.

There are a few examples of other forms of nervous disease in the family, which remain to be mentioned to complete this part of the family history. Five mothers among the men and one among the women were paralysed, and one died from disease of the brain. Two fathers died from apoplexy and one from paralysis. One maternal grandfather among the idiopathic males committed suicide while insane. Two cases of suicide as the result of drink have been mentioned in an earlier part of the paper. One woman was committed three times for attempted suicide, and made numerous other attempts while undergoing her imprisonment.

Table VIII. gives such details as I have been able to collect concerning crime on the part of other members of prisoners' families.

TABLE VIII.

TABLE showing the number of CRIMINAL RELATIVES of EPILEPTIC PRISONERS.

Relative.	IDIOPATHIC.			TRAUMATIC.	Net Total.	Remarks.
	Males.	Females.	Total.	Males.		
Father . . .	1	..	1	..	1	For drunkenness.
Mother . . .	1	..	1	..	1	"
Uncles . . .	3	..	3	..	3	{ 2 were Father's brothers.
Brothers . .	4	3	7	1	8	2 for drunkenness.
Sisters . . .	..	3	3	1	4	2 " "
Cousins . . .	2	..	2	..	2	{ Father's brothers' children.
Child . . . .	..	1	1	..	1	Female.
Total No. of Relatives convicted of Crime	11	7	18	2	20	

There are specially evident reasons for prisoners concealing any information they may possess on this subject. The knowledge of previous convictions so materially influences the result of any future criminal investigation, that they are naturally extremely reticent on the subject, both with regard to themselves and their relatives also. The difficulty is almost



as great in getting information from outside sources. There is, therefore, every reason for believing that the above table exhibits results considerably below the actual truth. The table of reformatory statistics before referred to gives on this point results greatly in excess of those for these older and epileptic criminals. Part of this may be due to children having a better knowledge of their relatives than adults who have been separated from home for many years; and part from their not appreciating the advisability of concealing the information.

TABLE IX.

TABLE showing the stated amount of CRIME amongst the RELATIVES of CHILDREN who have been committed to REFORMATORY SCHOOLS during the YEARS 1872 to 1876, inclusive.

Year.	No. of Boys.	Members of the Family who have been in Prison.					
		Father.	Per cent.	Mother.	Per cent.	Other Members.	Per cent.
1872	122	16	13·1	2	1·6	13	10·6
1873	154	15	9·7	4	2·5	23	14·9
1874	149	16	10·7	7	4·7	30	20·1
1875	150	19	12·6	3	2·0	43	28·6
1876	155	10	6·4	..	..	35	22·5

The only points noticeable in Table VIII. are that the collateral relatives are naturally the most numerous, and that drunkenness has been the offence in all the cases in which the nature of the crime has been ascertained.

Table X. (p. 514) shows briefly the general results obtained in the preceding pages with respect to the family history. The first part includes both direct and collateral relatives, the second part the former only.

From the first part of Table X. it will be seen that in the idiopathic cases drunkenness among the males and epilepsy among the females are respectively the forms of family taint most frequently met with, the very large proportion of epileptics among the latter making the net totals practically the same for both. There is also an increase both

TABLE X.

TABLE showing the GENERAL RESULTS of the INQUIRY into the FAMILY HISTORY of EPILEPTIC CRIMINALS.

	IDIOPATHIC.						TRAUMATIC.		Net Total.	
	Males.		Females.		Total.		Males.			
	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
With family his- tory of drink }	36	57·1	11	36·6	47	50·5	8	30·7	55	46·2
With family his- tory of fits }	29 <sup>1</sup>	46·0	21 <sup>2</sup>	70·0	50	53·7	4	15·3	54	45·3
With family his- tory of insanity }	16	25·4	9	30·0	25	26·8	3	11·5	28	23·5
With family his- tory of crime }	6	9·5	6	20·0	12	12·7	2	7·6	14	11·7
With direct here- ditary history of fits or insanity }	24	38·1	20	66·7	44	47·3	5	19·2	49	41·2
Without direct he- reditary history of fits or insanity }	39	61·9	10	33·3	49	52·7	21	80·8	70	58·8
With direct heredi- tary history of fits, insanity, drink, or crime }	47	74·6	21	70·0	68	73·1	9	34·6	77	64·7
Without direct he- reditary history of fits, insanity, drink, or crime }	16	25·4	9	30·0	25	26·9	17	65·4	42	35·3

<sup>1</sup> One had children only affected.<sup>2</sup> Two had children only affected.

of crime and insanity among the relatives of the females. This great preponderance of hereditary mental disease among the women as a predisposing cause of epilepsy and crime is seen perhaps better in the latter half of the table. Counting only direct relatives, 66·7 per cent. had one or more members affected with fits or insanity, as against 38·1 per cent. of the males. This result, I believe, accords with the statement usu-

ally made in speaking of hereditary insanity, that the female sex has a specially marked tendency to receive the taint from the parents. Classing together all possible predisposing causes, namely drunkenness, epilepsy, crime, and insanity, there is a difference of only 4 per cent. between the sexes. There is every reason to assume, judging from the conclusions arrived at by writers on mental disease, that if we could carry the family history of these criminals beyond the first generation, this amount of hereditary predisposition would be enormously increased. The amount of hereditary neurosis in these cases as they stand, however, is greatly in excess of that usually stated to exist by most authors. Dr. Reynolds found only 12 per cent. of epileptics giving a distinct history of epilepsy in other members of their family, and in only one-third of the total number of cases was there any nervous disease in either their direct or collateral relatives.

Hughlings-Jackson has "very little faith in the hereditariness of such symptoms as epilepsy or convulsions."<sup>1</sup> He cites a case where fits were known to exist in four members of one family, and remarks "that instances so striking are rare." Among criminals even more marked cases than this would appear to be far from uncommon. Since the above tables were made out, a man has been committed for trial, and is now in the hospital as a confirmed epileptic. The following is his hereditary history: Father had fits all his life, and died in one when prisoner was a child. He had been a very heavy drinker. Mother died 12 years ago from heart disease; was not subject to fits, and did not drink heavily. Has 6 brothers living; one is a heavy drinker, and epileptic, the others all healthy. Has had 4 sisters; 2 died from fits, aged 11 and 37 respectively. The latter was married, and had 6 children, all healthy. The two sisters living are both married, one has 6 and the other 7 children. In the elder sister's family one child has fits, and another died young in convulsions. An uncle—mother's brother—was subject to fits, and died from "fits and dropsy." Two of his own children had fits when young, and both are now dead. One cousin—a girl, æt. 14, daughter of another uncle, also on the maternal side—was in

<sup>1</sup> Reynolds's Syst. Med. vol. ii. p. 271.

an asylum, and died there from "decline." Prisoner himself has had fits all his life, and has been a heavy drinker for many years.

The question as to the relative amount of hereditary epilepsy among the different classes of society is doubtless a difficult one to answer. Dr. Reynolds is inclined to think that "hereditary taint is more frequently discoverable among the better conditions of life than among the poorer." I cannot help thinking that at the extreme lower end, at all events, the amount of hereditary nervous disease is much greater than is usually supposed. It may be true that the lower classes have become more habituated to such active determining causes as anxiety, alarm, and want; but it is probable that this would be more than counterbalanced by the increased amount of physical injury to which they are subject, to their long-continued habits of intemperance, and especially to the marked influence of hereditary alcoholism.

Passing now from the family history of these epileptics, it is interesting to inquire if there is any difference between them and ordinary prisoners in the character and amount of their crime, as evidenced by the number and nature of their convictions. I shall endeavour to be as brief as possible, but the question is important as furnishing corroborative evidence of many of the conclusions arrived at in the preceding pages.

Table X. gives, in a few well-defined groups, the number and nature of the offences for which these epileptics were committed to prison. Table XI. shows, under the same headings, the total committals for the year ending September 30th, 1876. I have selected 1876 for comparison, as a larger number of epileptics came under my observation during that year than in any other.

It should be remembered in connection with Table X. that committals to Wakefield Prison are alone referred to. Many epileptics gave a history of previous convictions in other gaols, but—for the better comparison of these cases with ordinary criminals—all such convictions have been omitted.

TABLE XI.

TABLE showing the NUMBER and NATURE of OFFENCES committed by EPILEPTIC PRISONERS.

Offences.	IDIOPATHIC.						TRAUMATIC.		Net Total.	
	Males.		Females.		Total.		Males.			
	No. of Con- victions.	Per cent.	No. of Con- victions.	Per cent.	No. of Con- victions.	Per cent.	No. of Con- victions.	Per cent.	No. of Con- victions.	Per cent.
Felony . . . . .	45	17·7	28	10·5	73	14·0	14	22·6	87	14·9
Rogues and Vagabonds and Vagrancy }	45	17·7	46	17·2	91	17·5	2	3·2	93	15·9
Neglect of Family .	2	0·8	..	..	2	0·4	..	..	2	0·3
Disorderly Paupers .	16	6·3	16	5·9	32	6·1	6	9·7	38	6·5
Common Prostitutes .	..	..	9	3·4	9	1·7	..	..	9	1·7
Assaults . . . . .	38	14·9	10	3·8	48	9·2	11	17·7	59	10·1
Wilful Damage . .	7	2·8	7	2·6	14	2·7	2	3·2	16	2·7
Drunkenness . . .	80	31·5	124	46·5	204	39·2	20	32·3	224	38·4
Bastardy Laws . .	8	3·2	..	..	8	1·5	..	..	8	1·5
Other Summary Con- victions . . . . }	13	5·1	27	10·1	40	7·7	7	11·3	47	8·0
Totals . .	254	100·0	267	100·0	521	100·0	62	100·0	583	100·0

Under the head of "Other Summary Convictions," in Tables XI. and XII., the epileptics are included one for each among the following offences. Fraud; obtaining living by dishonest means; non-payment of rates; hawking without a certificate; embezzlement; field robbing; keeping unregistered lodging-house; poaching; ill-treating a pony; resisting a constable; warehouse breaking; unlawful pawning; attempt at murder; travelling without a ticket; two for neglect of family; three for attempted suicide; and six for breach of bye-laws.

On comparing Tables XI. and XII., it will be observed, so far as the idiopathic males are concerned, that the proportionate

TABLE XII.

OFFENCES of PRISONERS committed during the YEAR ending  
SEPTEMBER 30th, 1876.

Offences.	Males.		Females.		Total.	
	No.	Per cent.	No.	Per cent.	No.	Per cent.
Felony . . . . .	1401	16·3	437	16·0	1,838	16·2
Rogues and Vagabonds } and Vagrancy	1141	13·3	213	7·9	1,354	11·9
Neglect of Family. .	167	1·9	3	0·1	170	1·5
Disorderly Paupers .	370	4·3	19	0·7	389	3·5
Common Prostitutes .	..	..	363	13·4	363	3·1
Assaults . . . . .	1059	12·3	119	4·4	1,178	10·4
Wilful Damage . .	247	2·8	56	2·0	303	2·7
Drunkenness . . .	2683	31·3	1074	39·4	3,757	33·2
Bastardy Laws . .	99	1·1	..	..	99	0·9
Other Summary Con- } victions	1439	16·7	441	16·1	1880	16·6
Total . . . . .	8606	100·0	2725	100·0	11,331	100·0

number of committals is greater among the epileptics for all offences except the following:—Neglect of family; wilful damage; and other summary convictions. For wilful damage the numbers are equal. An explanation for the increase among the vagrants and disorderly paupers has been already suggested. The percentage of committals for drunkenness is not appreciably greater. The year 1876 appears to have borne a higher percentage of committals than usual for this offence among the men. This is unfortunate, as there is reason to think that, as a rule, the proportionate percentage should be greater for epileptics than for non-epileptics. The last return I have by me, namely that for the quarter ending March 31st, 1878, gives the committals for drunkenness among the males at 24·6 per cent., or 7 per cent. below that of the epileptics. At the same time it must be remembered that drunkards are

often convicted of offences other than drunkenness, which have been committed simply under the influence of drink. The increased percentage under the heads of Felony and Assaults may owe its origin partly to this cause.

Turning our attention to the column for females, it will be observed that there is an increased percentage of committals among the epileptics for rogues and vagabonds, vagrancy, disorderly paupers, wilful damage, and drunkenness; and a decreased percentage for felony, common prostitutes, assaults, and other summary convictions. The increase in the first two classes of offences will probably be due to the cause suggested for the same fact in the male cases. With the increased percentage for drunkenness there is a decrease for assaults and felony. The total results for the idiopathic cases of both sexes show an increase for rogues and vagabonds, vagrancy, disorderly paupers, and drunkenness; and a decrease for felony, neglect of family, common prostitutes, and other summary convictions. The figures for wilful damage are the same. We might have expected to have found an increase for neglect of family; the reason for the decrease is, probably, that fewer of these prisoners are married, the existence of epilepsy not appearing to be a prepossessing feature. The number of committals among the traumatic males is too few to furnish any satisfactory conclusions when treated by themselves. The net totals for epileptics of all classes show an increase for rogues and vagabonds and vagrancy, disorderly paupers, drunkenness, and bastardy; and a decrease for felony, neglect of family, common prostitutes, assaults, and other summary convictions. The larger proportionate number of offences, then, for which these epileptics were committed appear to be those connected especially with drunkenness and vagrancy.

It has been asserted by Taquet that "sexual desires show themselves early in children of drunkards, and are associated with an absence of moral sense."<sup>1</sup> It is interesting, in connection with this statement, to observe that the percentage of convictions for bastardy is three times as great among the epileptics as it is among the non-epileptics. The interest of course lies in assuming that the epilepsy owes its origin to the

<sup>1</sup> Med. Record, Jan. 15, 1876. Page 8.

hereditary alcoholism which existed in all these prisoners. The above remarks, it must be remembered, apply only to the *comparative* number of convictions for the different offences. Inquiry into the *actual* number shows that not only are certain crimes more frequent among epileptics, but that the total number of convictions is considerably greater in all cases, both for males and females.

TABLE XIII.

TABLE showing AVERAGE NUMBER OF CONVICTIONS for EPILEPTIC PRISONERS.

	IDIOPATHIC.			TRAUMATIC.	Net Total.
	Males.	Females.	Total.	Males.	
Number of Prisoners . . . .	63	30	93	26	119
Total Number of Convictions	254	267	521	62	583
Average Number of Convictions	4·0	8·9	5·6	2·3	4·9

The difficulty in giving a similar table for ordinary offenders lies in the fact that in prison returns the number of *convictions* for any given time is stated, but not the number of *prisoners* convicted. One man, therefore, may be counted over three or four times, if he has been committed more than once during that period. I have specially examined the returns of about 2500 prisoners, discharged consecutively during the latter part of the year 1877, and eliminated this source of error so far as was practicable. The following are the results, it being premised that the average number of convictions given for each sex should, if anything, be lower than that stated:

	Males.	Females.
Total number of Prisoners . . . . .	2072	501
„ „ „ Convictions . . . . .	5957	2941
Average number of Convictions per Prisoner .	2·8	5·8

Prisoners convicted of drunkenness are, as a rule, sentenced



to short terms of imprisonment. The offence among chronic tipplers being frequently repeated, it follows that the number of previous committals for habitual drunkards would necessarily be in excess of that for other classes of prisoners. As there is little doubt about drunkenness being the more frequent offence among epileptics, it seemed advisable to discover, if possible, whether the increased average number of convictions for epileptics was due simply to the increased amount of drunkenness among them. It is impossible to determine the point accurately, on account of drunkards being so frequently committed for other offences. But I went carefully through the above returns for the females, as they are the chief offenders in this respect, with the following result:—Out of the 501 cases, 177 appeared to belong more especially to the drunken class. The total number of convictions for them was 1317, which would give an average per prisoner of 7·4, a considerable increase, it will be observed, on the numbers stated above, but still it does not reach that for the epileptics by about  $1\frac{1}{2}$ . If the drunken epileptics only were selected, the difference would be increased by at least twice as much again. It is impossible, therefore, to avoid the conclusion that the amount of crime, as indicated by the number of convictions, is greater among epileptics than ordinary criminals. The difference in the present series of cases is really very marked. The number of convictions for the 501 females was 2941; supposing these women to have had each the same average number of convictions as the epileptics, this total would have to be increased by 1517.

The same conclusion may be arrived at by comparing the number of first committals and recommittals. Table XIV. gives the particulars for the epileptics, and Table XV. for prisoners of all classes. It must still be remembered, with respect to the latter table, that if a prisoner has been recommitted during the quarter, he will have been counted more than once.

TABLE XIV.

TABLE showing the number of FIRST COMMITTALS and RECOMMITTALS of EPILEPTIC PRISONERS.

Previous Commitments.	IDIOPATHIC.						TRAUMATIC.		Net Total.	
	Males.		Females.		Total.		Males.			
	No.	Percent.	No.	Percent.	No.	Per cent.	No.	Percent.	No.	Percent.
Once . . . . .	12	29·3	5	23·8	17	27·4	5	41·7	22	29·7
Twice . . . . .	6	14·6	..	..	6	9·7	3	25·1	9	12·2
Thrice . . . . .	5	12·2	1	4·8	6	9·7	1	8·3	7	9·5
4 times . . . . .	3	7·3	1	4·8	4	6·4	1	8·3	5	6·7
5 times . . . . .	3	7·3	..	..	3	4·9	1	8·3	4	5·4
6 and 7 times . .	5	12·2	2	9·5	7	11·3	..	..	7	9·5
8, 9, and 10 times.	3	7·3	2	9·5	5	8·1	..	..	5	6·7
Above 10 times .	4	9·8	10	47·6	14	22·5	1	8·3	15	20·3
Total Recommitments	41	65·0	21	70·0	62	66·7	12	46·2	74	62·1
First Commitments .	22	35·0	9	30·0	31	33·3	14	53·8	45	37·9

The number of recidivists among the epileptics is greater by 16 per cent. and 13·1 per cent. respectively for the idiopathic and traumatic males, and by 5 per cent. for the females; the totals being an increase of 14·5 per cent. for all idiopathic cases, and of 9·9 per cent. for epileptics generally. In Table XIII. both the idiopathic and the net totals, compared with Table XIV., show a smaller percentage of prisoners with from 1 to 5 previous convictions, and a larger percentage for all numbers over 5. The total number of prisoners with more than 10 previous convictions is greater by 5 per cent. for the epileptics, but the increase, it will be observed, is due entirely to the females, who show an excess of no less than 18 per cent.

TABLE XV.

FIRST COMMITTALS and RECOMMITTALS of PRISONERS received during the QUARTER ending MARCH 31st, 1878.

Previous Committals.	Males.		Females.		Total.	
	No.	Per cent.	No.	Per cent.	No.	Per cent.
Once . . . . .	441	33·7	90	20·8	531	30·5
Twice . . . . .	210	16·0	41	9·5	251	14·4
Thrice . . . . .	154	11·8	39	9·0	193	11·1
4 times . . . . .	115	8·8	42	9·7	157	9·0
5 times . . . . .	73	5·6	24	5·6	97	5·6
6 and 7 times . . . . .	104	7·9	46	10·6	150	8·6
8, 9, and 10 times . . . .	71	5·4	21	4·9	92	5·3
Above 10 times . . . . .	142	10·8	129	29·9	271	15·5
Total Rcommitments . . .	1310	49·0	432	65·0	1742	52·2
First Committals . . . . .	1366	51·0	233	35·0	1599	47·8

Of the 10 idiopathic females with more than 10 previous convictions:—

1 was committed 51 times, including 26 for drunkenness.

1	"	36	"	26	"
1	"	27	"	5	"
1	"	18	"	12	"
1	"	18	"	10	"
1	"	14	"	7	"
1	"	13	"	3	"
1	"	12	"	9	"
1	"	12 times,	"	all for drunkenness.	
1	"	17 times.			

Of the 4 idiopathic males:—

1 was committed 30 times, including 21 for drunkenness.

1	"	22	"	7	"
1	"	12	"	3	"
1	"	11 times.			

The 1 traumatic male:—

was committed 14 times, including 8 for drunkenness.

These 15 cases have an average of 24 convictions each, including 9·1 for drunkenness.

In Table XVI. are given the details of the habits of the prisoners and their fathers in the case of epileptic and non-epileptic males. The figures for the latter are part of the table before referred to as having reference to prisoners committed for 6 months and over.

TABLE XVI.

TABLE showing the HABITS of the PRISONERS and their FATHERS in the Case of EPILEPTIC and NON-EPILEPTIC MALES.

	Epileptic.	Non-Epileptic.
	per cent.	per cent.
Number of Prisoners of intemperate habits . . .	75·7	66·0
Fathers drunkards . . .	67·8	51·5
„ sober . . . . .	32·2	48 5
Number of Prisoners of temperate habits. . . .	24·3	34·0
Fathers drunkards . . .	66·6	30·9
„ sober . . . . .	33·4	69·1

I do not wish to lay too much stress on these figures, as the records of the epileptics are somewhat imperfect on this point, and also because the two classes of prisoners are not strictly of the same criminal type; but the table seems to lead to the following general conclusions:—

- (1) There are more drunkards among the epileptics than among the non-epileptics, the proportion of temperate to intemperate prisoners among the latter being as 2 to 1, and among the former as 3 to 1.
- (2) Among the non-epileptics, the percentage of drunken fathers is greater for prisoners of intemperate habits, and the percentage of sober fathers greater for prisoners of temperate habits.
- (3) Among the epileptics, the percentage of drunken and sober fathers is practically the same for temperate and intemperate prisoners.
- (4) The percentage of drunken fathers among the epileptics is greater, both for temperate and intemperate prisoners, than it is for the intemperate non-epileptics.

With regard to the epileptic females, the case is a little different; the number of drunkards among them is even still greater than among the men. It is the exception to meet with an epileptic female criminal who is not at the same time intemperate. Among these intemperate epileptic women the percentage of sober fathers is greater than the percentage of drunken fathers. It is probable that a more perfect acquaintance with their family history would show a larger proportion of drunkards among the mothers, but, making allowance for this, there is little doubt that the amount of hereditary alcoholism is considerably less with the women than it is with the men. On the other hand, we find that the amount of nervous and mental disease in the family is greater by at least 20 per cent. Whereas, then, epilepsy in men is associated especially with alcoholism in the parents, in women it is found more frequently in connection with epilepsy and insanity in other members of the family.

TABLE XVII.

TABLE showing the Number of EPILEPTIC PRISONERS suffering from other Forms of NERVOUS or MENTAL DISEASE.

DISEASE.	IDIOPATHIC.				TRAUMATIC.	
	Males.		Females.		Males.	
	No.	Per cent.	No.	Per cent.	No.	Per cent.
Confined in Asylum . . . . .	3	4·7	4	13·3	2	7·6
Demented or Imbecile . . . . .	2	3·0	..	..	1	3·8
Hemiplegic . . . . .	8	12·7	1	3·3	2	7·6
With Facial Paralysis . . . . .	2	3·1	..	..	..	3·8
Cripple from Birth . . . . .	1	1·5	..	..	..	..
Deaf and Dumb . . . . .	..	..	1	3·3	..	..
Wasting of limbs from Infantile Paralysis. . . . .	..	..	1	3·3	..	..

It is further noticeable, that the number of convictions for the intemperate epileptics of both sexes is greater than for the

temperate; the excess being due to the increased committals for drunkenness, and, as one would naturally expect from the foregoing, affecting the women to a much greater extent than the men.

In addition to their epileptic convulsions and frequent drunken habits, several of these prisoners were affected with other forms of nervous disease. (See Table XVII., p. 525.)

TABLE XVIII.

	CASES.											Summary.
	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	
Sex (M. Male, F. Female)	M.	M.	M.	M.	M.	M.	M.	M.	M.	M.	F.	10 M., 1 F.
Age . . . . .	60	50	53	33	39	25	42	76	24	23	33	
Class (I. Idiopathic, T. Traumatic) . . . }	I.	I.	T.	I.	I.	I.	I.	I.	T.	I.	I.	9 I., 2 T.
Duration of Fits in years	24	22	15	33	8	25	3	76	2	15	4	
Duration of Hemiplegia in years . . . }	24	22	10	(?)	8	25	3	2	$\frac{1}{2}$	5	(?)	
Side affected (R. Right, L. Left) . . . }	L.	L.	R.	L.	L.	R.	L.	L.	R.	R.	L.	7 L., 4 R.
Habits of Prisoner (D. Drunkard, S. Sober) }	D.	S.	S.	S.	..	..	D.	S.	D.	..	..	3 D., 4 S.
Habits of Father . . .	D.	D.	S.	D.	S.	(?)	D.	S.	D.	S.	S.	5. D., 5 S.
Habits of Mother. . .	..	..	D.	..	..	..	..	..	..	..	..	1 D.
Other members of family } Drunkards . . . }	..	..	..	1	..	..	..	..	..	..	..	
Total No. of Convictions	30	6	1	2	5	1	1	8	1	1	1	
No. of Convictions for } Drunkenness . . }	21	..	..	..	1	..	..	..	..	..	1	
No. of Relatives with } Fits . . . . . }	1	..	..	6	2	..	..	2	2	..	..	
No. of Relatives Insane	..	..	..	1	2	..	1	1	2	..	..	

Of the 9 who had been insane, 3 men and 3 women had been confined in an asylum once, 1 man twice, 1 woman three times, and 1 man four times. In addition to the above cases of distinct mental disease, many of these epileptics are

more or less weak-minded, being dirty in their habits and idle at their work; they are occasionally given to emotional outbursts, and exhibit a general want of moral self-control. The existence of so large a number of cases of paralysis is somewhat remarkable. The ordinary temporary hemiplegia following an epileptic fit is not here referred to. All the 11 prisoners in the above table had organic hemiplegia at the time they came under observation. It is further remarkable that, in the large majority of cases, 7 out of 9 among those returned as idiopathic, the hemiplegia was on the left side. The principal details in connection with these cases may be gathered from the tabular analysis opposite (Table XVIII.).

## Critical Digests and Notices of Books.

*A Defence of Philosophical Doubt, being an Essay on the Foundations of Belief.* By ARTHUR JAMES BALFOUR, M.A., M.P.  
London : Macmillan and Co., 1879. 8vo. pp. 355.

THE author announces in his Preface that his work "may be sufficiently described by saying that it is a piece of destructive criticism formed by a series of arguments of a highly abstract character,"—an awful announcement, as he seems to think, and as deterrent as if it had run, "Who enters here must leave Belief behind." When we do enter, however, into this cave of doubt, it is found to be not quite so dark as the threat implies—the possibility of belief up to a certain practical point being left as a kind of residuum of the destructive analysis ; so that, notwithstanding that absolute certainty about anything is unattainable, a healthy man may yet eat his dinner in the reasonable expectation of digesting it. The philosophic question raised is whether beyond the limits of observation the universe is under the same reign of law as we observe it to be within them. It may, or it may not be—and this "may not" supplies the main force of this critical attack.

The only possible foundation of science being the belief in the law of universal causation, and this fundamental law itself being only provable from particulars by observation and experiment, it follows that if particulars which are beyond the range of observation and experiment may contradict the law, the universal truth of any scientific fact cannot be assumed to be absolutely free from a kind of doubt, which one may if one pleases call philosophical.

There may be no real doubt in the mind of any student of science that the law of causation is universal, but the possi-



bility of imagining doubt must, we think, be admitted to have been shown by our author. And this distinction leads us to ask what he means by philosophic belief and philosophic doubt? Something like an explanation is found at page 5, where the author draws what he considers a most important distinction between the causes which produce a belief (or doubt?) and the grounds which justify one. The causes, he says, are psychological, the grounds or reasons are philosophical. The author admits that "in constructing a philosophy a previous psychological enquiry may be required," but he denies that the causes of belief include the grounds for it, and that psychology can comprehend philosophy and all other mental actions. The science of mind may prepare the ground for a philosophy, but cannot, he says, add one fragment to its structure. That, however, must depend upon what is called the science of mind, or psychology. If it includes the knowledge of mental powers and aptitudes, the assertion is incorrect, and the error has an important bearing on the criticism on Mill, whose logic is adapted to mind as it exists with limitations of experience; whereas the demands of the author are founded upon mind as it may be conceived without limitations of experience and of knowledge. As a philosophical exercitation the criticism is successful, as showing that Mill's system does not and cannot prove the absolute and entire truth about Nature, that is, the Universe in all its conceivable extension in time and space; that it cannot even prove the universal existence of the law of causation or of uniform succession, much less that of any of the subsidiary laws dependent thereupon. But what then? It shows that man, not being omniscient, may perhaps conceive a philosophy founded upon the assumption that he is so, but which in his present state must remain a dream of the imagination, like the cap of wisdom and the sword of sharpness, and the three-league boots of Jack the Giant Killer.

In the meantime the author comforts us with the assurance that

"In all cases of Induction we can do no more than prove a certain law to be *probable*. If our observations or experiments be numerous and successful, the probability proved may be a very

high one; if they are few and ambiguous, it may be a very slight one; but in either case, what we prove is probability, and probability alone. This, however, need cause us no uneasiness. If demonstrative certainty is denied us, we may still by this method obtain that practical certainty which is all we require to guide us in the affairs of life."

This also seems the opinion of Professor Jevons, and, after all, the *laches* of which Mr. Mill appears to be convicted are, that by avoiding to state the philosophical shortcomings of the method of proof from particulars, he has so stated his position that it cannot in reality "for a moment stand against hostile criticism." Considering what Mill has done to improve the method of ratiocination which enables us to "attain all that practical certainty which is all we require to guide us in the affairs of life," the judgment does seem heavy for the offence.

The author supposes himself to be asked whether the existence of a persistent universe, governed by causation, really does require proof, seeing that the belief in such an universe lies at the root of all knowledge, and the proof of it is impossible and unnecessary; and he replies that, while as a matter of fact, the law of causation does not appear to be accepted in its integrity by the greater part of the human race, and that those who do accept it seem to feel the necessity of founding it upon some kind of proof; with regard to the persistent universe, practically every one does believe in it, even those who speculatively question it. But is it possible to believe in a persistent universe without that belief which seems necessarily complementary to it, in the constant succession of events, which is the law of causation, and the foundation of all law? Persistent universe without law seems inconceivable.

The objection, from the occurrence of optical or other so-called illusions of the senses, which the author takes to the immediate belief which he admits that the majority of mankind certainly have in the testimony of their perceptions, seems to us forced and untenable. Granted that this evidence is sometimes untrustworthy, and that perceptions may arise in us which have no correspondent objects, it is of the very essence of the method of induction to eliminate such sources of error; and an inductive philosophy of perceptions will

not confuse those which are false and illusory with those which will bear the tests of verification. It is curious that after taking this objection to a knowledge of the universe, founded upon illusions of the senses, the author should be able so clearly to state the great difference between thought which has a true relation to sense, and thought which has not. "Grant that everything which is real is thought, it cannot be the fact that everything which is thought is real, since if it were so, mistakes as to the true nature of any object would be impossible; a doctrine as subversive of science as any form of idealism ever devised."

The obvious answer to this paradox is that everything which is thought is real as thought, and that the examination of all thought, even of that which has no known correspondence with outside causes of thought, is a proper and by no means subversive subject of mental science.

The middle chapters are devoted to the Transcendentalism of Kant and the Authority of Consciousness of Hamilton; but it is hard to understand the necessity for discussing these metaphysics in relation to scientific beliefs. The following three chapters, in which Mr. Herbert Spencer is belaboured, keep closer to the professed object of the work, although they come more near than need be to a general and unfavourable criticism of Mr. Herbert Spencer's opinions; the conclusion of which is that although Mr. Spencer has an astonishing faculty for collecting from every department of knowledge the facts which tell in his favour, his sensibility is blunted in the matter of difficulties and contradictions, and on metaphysical ground he is a trespasser. It cannot, however, be denied that in the Inconceivability of the negative Mr. Spencer has supplied just that axiomatic test of certitude which is demanded by philosophical doubters or debaters, a test whose main value may be that of an intellectual gag, but as such firm and efficient. At the twelfth chapter we somewhat escape from the torrent of destructive criticism, and are allowed to set foot on the solidifying ground of the author's own opinions. Considering science as a logical system, he tells us that "among bodies scattered through space is assigned to each man one of especial importance to himself, his own organism." "The

peculiarity about them [these organisms] is that they are the immediate channels of connection between the world I have just described and the thinking beings who by their means are made acquainted directly with the appearance of that world, and indirectly with its nature and constitution," p. 243. This description by one who shares the "implicit and indestructible confidence in the truth of that which had to be proved" by science, is not a little remarkable, and leads to surmise as to what the author's psychology may be, which induces conviction in the overwhelming scientific evidence of the truth that "all beliefs are the result of the operation of natural causes, and of these alone," p. 274, and in that "physiology which shows it (the evolution of belief), *à priori*, by demonstrating the dependence of thought upon the organism, and of the organism in inheritance and environment," p. 261. It was possible that the author might maintain that all beliefs are the result of natural laws acting upon the organic channels of communication which exist between the world, that is the environment, and of the thinking beings which are at the other end of the channels; but by ordinary persons the evolution of belief by natural and physiological processes would seem to be inconsistent with power of regarding each man's own organism merely as a channel of communication between the world and the thinking being, that is to say, between the environment and his real self.

It must be over subtle or merely fanciful to say that scientific notions are anthropomorphic in the same sense that our ideas of the Deity are so, and if the former the point is too fine to be felt. The idea of inorganic force has sometimes been very clumsily likened to that of animal effort, but it would be difficult to say what other scientific notions can by any straining of imagination be called anthropomorphic. Yet this is the leading argument in one of the author's most intelligible chapters—that on Science as a Logical System. Another objection to belief in our perceptions is that, while some of our observations of the qualities of bodies are apparently true, as that of extension, others, as that of the quality of colour, are not true; bodies being themselves devoid of colour, which resides in the vibrating medium and

the organs. Great capital is made out of this, which is merely a bit of slipshod wordsigning. If it be not said that a violet is blue, but that a violet is bluecausing, the statement would be as scientifically accurate as that a violet is soft and small, and all the philosophic fog raised by the author about the untrustworthiness of scientific observation about coloured bodies would disperse. Moreover Science, and Science alone, does provide us with means of distinguishing perceptions which are immediate from those which are mediate or altered, and those which have an object from those which are illusory; to distinguish the author's moonshine, for instance, to which he makes a special reference at page 255, from the brain-begotten vision of a ghost, which he calls *optical* delusion.

The thirteenth chapter, on the Evolution of Belief, stands in great and remarkable contrast to the remainder of the volume, and the author has felt and excused the inconsistency. It would be impossible to give a more full and frank adhesion to the great doctrine that "all beliefs are the result of the operation of natural causes and of these alone" than the author has here done. "The scientific evidence for this truth (the evolution of belief) is various and overwhelming;" and it is much for the author to admit that this can be said of any scientific evidence. The argument in favour of anti-scientific scepticism drawn from this law of mind is obvious enough. Belief having been evolved from the influences of heredity, environment, and other circumstances, we have reason to expect that it will continue to evolve,—that is to say, to change; and seeing that we have reason to think that the evolution hitherto has been in the direction of truth, we must expect future change to be towards greater truth than we at present possess, which is an admission that what we now believe is true only in part. This argument and its conclusion will, we think, be gladly accepted by those against whom it is aimed, the men of science, for, unlike the philosopher who demands absolute truth in order that he may gain "repose of mind," they covet the changes of extending knowledge, and hoping for nothing more than relative truth, they labour to obtain its continual increase. But it is to be remarked, that although all beliefs result from natural causes, they are not, therefore,

as the author supposes, all within the stream of evolution. There is no reason to expect that any beliefs which can properly be called fundamental, as the belief in one's own existence, will ever change. This, it would seem, must have been the same in kind in the first conscious being as it is now, and, as it would seem, must remain through all the æons of change which may ensue.

We will not follow the author in his latter pages, in which he compares what he calls the two creeds of Science and Religion, and justifies himself for attacking the one because it is, he says, opposed to the other. Should any one wish to avail himself, for a similar purpose, of the weapons he supplies, the best way will be to imitate many novel readers, and commence at the end of the book, which, like the end of a whip, constitutes the lash. Science will perhaps outlive all this destructive criticism, and the humble workers in the pursuit of knowledge who do not uphold any aggressive creed for the purpose of undermining the foundations of faith may take courage from the author's admission made at p. 280: "It is conceded that similar effects always have similar causes, and that a knowledge of particular sequences and co-existences between phenomena can be derived from observation."

The general effect of the book reminds one of the following dialogue in Congreve's 'The Way of the World':

*Petulant.* "If he says black's black, if I have a humour to say 'tis blue—let that pass—all's one for that. If I have a humour to prove it, it must be granted."

*Whitwoud.* "Not positively must—but it may—it may."

*Petulant.* "Yes, it positively must, upon Proof positive."

*Whitwoud.* "Ay, upon Proof positive it must; but upon Proof presumptive it only may, that's a logical distinction now—Madam."

*Mrs. Marwood.* "I perceive your debates are of importance, and very learnedly handled."

*Petulant.* "Importance is one thing, and learning's another; but a debate's a debate, that I assert."

The degree of proof demanded by our author is Petulant's proof positive, while poor Mr. Mill and others, who diligently hunt the footsteps of Creation, can only with difficulty attain

to proof presumptive. We are glad to find, however, that Mr. Balfour, although he seems quite disposed to expect that others will consider his "piece of criticism so purely destructive in character," more like "a mere dialectical puzzle, a mere exercise in ingenious objections, or even a contribution of a somewhat eccentric kind towards English philosophy," than a serious estimate of the Foundations of Belief, vouches for his own earnestness. The importance and learning of his work may be questioned; but a debate's a debate, and this debate proves with certitude that the author believes in it himself.

JOHN CHARLES BUCKNILL.

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*The Relations of Mind and Brain.* By HENRY CALDERWOOD, LL.D., Professor of Moral Philosophy, University of Edinburgh. London: Macmillan and Co., 1879.

THERE are some who have a vague belief that Physiology is going to supersede Metaphysics, or that all inquiries as to the mode and action of the mind are shrouded in the darkness of the unknowable, and that the utmost we can learn of mental operations consists in ascertaining the swiftness of thought, or the time between a sensation and the answering motion calculated by the help of some delicate chronometrical instrument. But questions connected with the nature and destiny of the human mind are too deeply interwoven with man's dearest hopes and feelings to cease to have an interest for an active and inquiring intellect. Unlike most men who have written on the physiology of the brain, Dr. Calderwood began as a student of Mental Philosophy, and then became a student of Anatomy and Physiology. His book is interesting, not only to the physiologist and neurologist, but also to the philosopher who is anxious to study the relation of feeling and thought to the nervous system.

Dr. Calderwood's reputation as a metaphysician is already established, and in his book he shows that he has mastered what is known of the general anatomy and physiology of the

nervous system down to the very latest researches into the functions of the brain. In his first chapter, "On the Relation of Philosophy and Science," Dr. Calderwood rebuts the accusation made against the Scotch school of philosophy of constructing their theories in disregard of observation. "Those," he writes, "who would fasten upon psychologists of former days such a charge as this must be prepared to maintain that thought and recollection and volition are not facts, or that these are facts which do not occur according to law, or that both facts and laws are beyond the reach of scientific inquiry." Dr. Calderwood has little difficulty in showing the fallacy of Dr. Maudsley's attack on the trustworthiness of consciousness. "It has no doubt been said that there 'is no agreement' among those who resort to internal observation, but every one who is familiar with the works of psychologists knows that the statement is inaccurate. As to the laws of observation, of association, of reasoning, of pleasurable feeling, there is all but perfect agreement among them." And again: "It were indeed strange were any science to offer itself as defender of the dogma—that man is not to trust his own experience. What may he trust if not this? Take the most ordinary and commonplace tests. How but by consciousness does any man know that the blue appearance of his hands on a wintry morning is the accompaniment of coldness, or that perspiration is attendant on a high degree of heat? When a man says that he feels warm, is it a sad misfortune that 'he appeals to a witness whose evidence can be taken by no one but himself?' Is it true that this is a witness 'whose veracity cannot be tested?' To throw doubt upon experience is to deny the possibility of knowledge, and so of all science. When the psychologist trusts his consciousness he simply does what every man does—what every man must do, who is not insane. Trust in consciousness is no speciality of psychologists, but the common prerogative of humanity."

Dr. Calderwood then proceeds, in four chapters, comprising about 150 pages, to give a sketch of the comparative anatomy of the brain and nervous system, and to discuss what is known of their functions. These chapters, though of course compilations, are written in a shrewd and critical vein, which displays



the author's vigorous train of thought. Working with the assistance of such distinguished scientific men as Professor Turner, Dr. Rutherford, Dr. Grainger Stewart, and Dr. Clouston, this part of the work may be regarded as a trustworthy *résumé* of the newest results of modern investigation into the functions of the nervous centres, written in a style which renders it of easy understanding to any cultivated reader. The descriptive portion of the book is illustrated by twenty-eight engravings on wood, taken principally from published works.

In Chapter V. he compares the brain and intelligence of man with those of some of the higher animals, especially the dog, horse, ape, and elephant. Dr. Calderwood places the intelligence of the dog much higher than any of the others, and quotes authorities to show that the sagacity of the elephant has been much overrated. He shows that while in the anthropoid ape there is a near approximation to the human brain, the creature does not show intelligence equal to that observed in the ordinary life of animals, much less similar in the structure of the encephalon.

"The ape is far less like the man in intelligence than its brain is like to the human brain." He reaches the conclusion that the brains most elaborate in convolution are associated with the most highly developed muscular system, and finds in the similarity of the body in the ape the explanation of the similarity of its brain.

Dr. Calderwood does not seem, at p. 206, to distinguish nicely between complexity of the muscular system admitting of diversity of execution, and mere mass of muscles allowing for great exertion of force. The brain of the horse, the elephant, or the whale, is much more finely convoluted than that of the cat; but yet the cat is assuredly much more capable of fine and difficult motions than the horse or whale; and although the elephant is an animal of colossal stature, its strength seems to me to be feeble in comparison with its bulk. Dr. Calderwood might advantageously have considered the case of the seal, an animal with a well-developed brain and possessing a good amount of intelligence, but very limited power of muscular exertion.

In general, Dr. Calderwood takes a much lower view of the

intelligence of animals than naturalists who think that the human intellect has been evolved by a series of improvements from that of the brute.

He doubts whether animals can exercise any voluntary determination over their actions, and thinks that most things that they do can be accounted for by sensori-motor activity. Dr. Calderwood keeps clear of the question whether the mental manifestations of the higher vertebrate animals are to be accounted for by the hypothesis of a supersensuous principle, or are simply a form of nervous activity. However low we may rate the amount of reason in the lower animals, most people will be disposed to admit that if what thought exists in the dog is but a function of the brain, even the highest abstract thought might be a function of the brain in man. Hence naturalists like Agassiz, who believe that there is a soul in man, admit the existence of a similar immaterial principle in animals. We should like to have Dr. Calderwood's opinion on this point, but the learned professor is too skilful a dialectician to give battle save on ground selected by himself.

When we come to Chapter VII. "On Personal Experience as connected with Sensation," we feel the advantage of Dr. Calderwood's skill as a metaphysician. He does not contest that molecular changes accompany sensation, motor impulses and thought, but he shows that there is no logical connection recognised by the mind between motions in cells and thought and sensation. "All that we know of nerve-substance," he writes, "whether fibre or cell, fails to explain the simplest and most familiar fact in our experience." Not only have we a series of sensations, but we recognise this series, and assume a personality which has experienced those that are past.

Dr. Calderwood makes use of the argument so well handled by Huber, in his tract, 'Ueber das Gedächtniss:' "Even if," he observes, "physiological hypothesis were ventured in the form of a suggestion that there may be in the sensory cell a *register* of the shock delivered there, this would not help us towards an explanation of the facts of consciousness. Even if there were such a register, and the registration were made on a sensitive surface, and were permanent, this would not meet the requirements of the case. A register contains the mate-

rials for comparison, but does not institute comparisons. The facts carry us quite beyond mechanical contrivance, inasmuch as one thing not only follows another, but one thing is compared with another; that is, there is not only one thing distinct from another, but one thing is distinguished from another."

Dr. Calderwood takes up a familiar calculation of so many cells to the square inch, and gravely argues that there is not a sufficient number of cells for the vast number of ideas which we acquire. We have always considered this sort of thing so much solemn trifling; as Spinoza observes we cannot conceive of a space bounding an idea, or an idea bounding a space. Could a big nerve-cell hold more ideas than a small one? Or does a great idea ever smash a frail one, if it comes to a collision in a nerve-fibre?

Dr. Calderwood goes on mingling metaphysics with physiology and cerebral pathology, as far as they may be mingled. He treats of the use of speech, the action and reaction of mind and body, sleep and unconsciousness. He tries to show how the observations of physicians in insanity do not invalidate the belief in the independent existence of the human mind. This part of his argument is treated with great care, and shows considerable reading and fulness of information.

It is no doubt the duty of a critic to pick out a number of faults and oversights in the book, and I am concerned to think how carelessly this office must have been performed, for I cannot recall any salient blunders, and am not willing to read the book over again. Now and then there is a certain simplicity noticed which bespeaks the amateur, as when Dr. Calderwood quotes Carpenter's 'Physiology,' to prove the fact familiar to every surgeon, that a severed nerve may be reunited, and the power of communication restored; or when he speaks of checking brain-wasting, and reinvigorating the organ with "such things" as tincture of opium and sulphuric ether.

Dr. Calderwood's idea that we may have loss of a portion of brain-substance with advantage to the mental powers is perhaps original; not so his suggestion that the brain development may so far naturally exceed the capacity of the cranium as to cause compression of the whole brain. His medical

friends will be able to put in his hand some account of the disease called hypertrophy of the brain.

In his concluding Chapter, "On the Higher Forms of Mental Activity," Dr. Calderwood lays great stress upon the existence of a sense of duty in man, and a sense of dependence upon a higher power which he thinks could not have originated from any organisation of matter; but for this and other arguments the reader must turn to the book itself.

Enough has been said to show the character of the work. The subject must be an interesting one to all who occupy themselves with mental philosophy, and it is treated in a manner worthy of its importance.

WILLIAM W. IRELAND.

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*Müller on the Acute Anterior Poliomyelitis of the Adult. (Die Acute Atrophische Spinallähmung der Erwachsenen; eine klinische Studie.)* By DR. F. MÜLLER. (Pages 105.) 8vo. Stuttgart: Enke, 1880.

FEW clinical types have been more diligently and successfully studied during the last decade than that originating from an inflammation, acute or chronic, of the anterior horn of spinal grey matter. "Poliomyelitic" diseases comprise the "essential" or infantile paralysis of the older authors, the (acute or chronic) atrophic spinal paralysis of the adult, bulbar paralysis, and some forms at least of progressive muscular atrophy. The name "regressive paralysis," which has been proposed, seems unfortunate, for though expressive enough of some characteristic features of the acute forms, it certainly fails to apply to the subacute and chronic forms.

The first author who seems to have had a clear idea of the "infantile" type is Underwood (1784), who in his work on diseases of children describes it as paralysis "of dentition." Kennedy (Dub. Med. Jour. 1841) speaks of a "temporary" paralysis of children, and the name "essential" was given to the disease by Barthez and Rilliet. It was with Heine and

Duchenne (1850–1860) that the idea of a spinal localisation arose, and their theoretical assumption has since been verified by numerous autopsies.

The clinical characteristics of the infantile disease, as delineated by Duchenne, were so striking as not to fail being recognised in a number of adult cases; and it was about ten years ago that the indefatigable author of the ‘*Electrisation Localisée*,’ described a “*Paralysie spinale antérieure aiguë de l’adulte, ou par atrophie des cellules antérieures.*” Dr. Müller, in the able and exhaustive monograph, gives us a careful *résumé* of all the cases published from Duchenne’s to the present day, and adds four observations of his own; one of them quite unique, owing to the fact that the patient was under observation from the very beginning of the disease; and that a most thorough electrical exploration of nerves and muscles was carried out from the very onslaught of the paralytical symptoms daily during the first week, and at appropriate intervals during eighteen months subsequently.

The author holds that *fever* is present in all cases as an initial symptom. It can be only a “*lapsus calami*” of Eulenburg’s, when he says that “fever has hitherto not been observed” in the disease (*Lehrbuch*, vol. ii. p. 378). From the cases hitherto published it would appear that the duration of febrile disturbance is usually from two to seven days. The intensity of the fever varies much, but bears no proportion to the extent of the subsequent paralysis.

*Pain* is a very common symptom during the first stage of the disease, and is usually of a tearing character; but, like fever, varies much as to duration and intensity. It extends over the limbs, the sacrum, or along the vertebræ. It disappears with the invasion of the paralysis. It must be noted, however, that later on other algæic symptoms may supervene, which, however, are duller in character and more localised in the muscles undergoing atrophy.

Various *paræsthesiæ* are apt to make their appearance; but the presence of any anæsthesiæ at once relegates the case from the poliomyelitic group to that of the more diffuse myelitis, where the posterior tracts of the case are involved; as, for instance, in the case described by Glynn (*‘Lancet,’* 1878,

vol. ii. p. 395), where there occurred a complete anæsthesia of the four extremities, lasting two months.

The leading symptom, *loss of motor power*, occurs as a rule abruptly, not gradually. Whereas in the infantile form monoplegic lesions are the most common, in the adult the four extremities are most usually affected; each occurring in about 50 per cent. of the respective cases. An interesting point, to which I shall have the opportunity of referring more fully elsewhere, is that the paralysis does not distribute itself among muscles according to their peripheral nervous supply, but rather according to their central nuclear connections. No muscle can claim immunity from poliomyelitic processes, though some writers have expressed themselves in this sense. Both tendon and skin *reflexes* are, as a rule, abolished or impaired, but reappear in the regressive stage, though apparently without keeping pace with the rate of recovery of the muscular power.

In typical cases of poliomyelitis, no vesical, nor rectal, nor genital disturbances occur. On the other hand, *vaso-motor* changes are always present. Dr. Müller found in his case that the lividity and coldness of the affected extremities which characterise the later stages, are preceded during the first few days by a hyperæmic and hyperthermic condition.

From the fact that disturbances in the functions of the sweat glands are but rarely observed in the paralysed limbs, Dr. Müller concludes that their functions are independent of the vaso-motor apparatus, which is always impaired; and that the nervous centres which govern them are not situated in the motor tract of the anterior horns.

The only *trophic* troubles present in poliomyelitis consist in degenerative changes of nerve and muscle, as evidenced by the frequent wasting of the limb and by the electrical reactions obtained. The latter have been, as before mentioned, most fully investigated in one of Dr. Müller's cases; and his results concord most satisfactorily with the phenomena of the "reaction of degeneration," so ably described by Professor Erb in vols. xi. and xii. of Ziemssen's 'Cyclopædia.' The facts may be summarised as follows: The farado-nervous excitability disappears rapidly; in the most severely affected nerves

within five or six days of the paralysis. The time of its reappearance varies much, and is always preceded by that of voluntary stimulation. In permanently disabled nerves it, of course, vanishes for ever.

The galvano-nervous excitability follows closely the behaviour of the latter, but takes about two days longer to disappear. Its restoration also follows that of voluntary power.

There are frequently a number of nerves which, though the muscles supplied by them may undergo atrophy, and all the changes of reaction dependent upon it, never show any, or but a slight, diminution in farado-galvanic excitability. Cases where this occurs form an interesting transitional group, characterised by a "partial" (or "middle") form of reaction of degeneration, which shows a disturbance in the spinal influence on the nutrition of the muscle only, and not of its supplying nerve.

With regard to muscles, their farado-excitability diminishes more slowly than that of the nerves, but eventually disappears completely. But the most interesting changes are those offered by the galvano-muscular reactions. After undergoing a slight diminution (which in Dr. Müller's case lasted about three weeks), the galvano-muscular excitability rises, and remains above the normal for a few days; then it gradually diminishes again, whilst the pathognomonic alterations in polar effects manifest themselves. These, in severely affected muscles, amount to a complete inversion of the normal polar formula:  $A.C.C. > C.C.C.$ , and  $C.O.C. > A.O.C.$  In muscles whose degeneration is less marked, we get  $A.C.C. = C.C.C.$ , or simply a slight rise of the anodal effect, without any corresponding cathodal increase. At the same time the mechanical irritability of muscles increases, and the contractions are altered in character, becoming protracted, sluggish, vermiform. These alterations are of great prognostic importance. Muscles in which recovery is never to take place, have a uniform downward progress; stronger and stronger currents are necessary to obtain the trace of a contraction, the "ultimum moriens" being the A.C.C. In less severely affected muscles the qualitative alterations of polar reactions are not so marked at first,

and gradually return to the normal formula as the degenerative process enters into a regressive stage. Of course the partial form of degenerative reaction, viz. where the nerve does not share in the morbid process, is of far better augury as to the eventual recovery.

An interesting fact is mentioned by Dr. Müller. The paralysed right rectus abdominis showed no quantitative nor qualitative departure from the normal reactions; apparently a contradictory result. I might suggest as an explanation that, as in some forms of peripheral paralysis (such as mild cases of facial or musculo-spiral paralyses), where the electrical reactions remain normal throughout, the transmission of voluntary impulses alone is interfered with by the lesion, whilst that of trophic influence is left intact. This is made all the more admissible by the well-known facts that in mixed nerves sensibility resists influences destructive of motility; and that, as we have just seen, muscle-nutrition may be affected without any participation of the nerve in the morbid process.

The *regressive stage* of the disease is inaugurated by the restoration of those muscles in which mere quantitative electrical changes have been observed. This may begin as early as within a week of the paralysis; or may be delayed for two or more months when the lesion is more severe. The atrophy of the muscles may be masked by deposition of adipose tissue. As a rule, residual paralyses persist here and there, and secondary contractures may supervene. With regard to the *morbid anatomy* of the acute poliomyelitis of the adult, it is only since Schultze published his case (Virchow's 'Archiv,' 1878), that a complete answer could be given to those who objected to the localisation of the disease in the anterior horns. The patient, aged 40, died twenty months after the attack.

Macroscopically, two spots of the anterior horns in the left cervical and right lumbar enlargement were found discoloured; and the horn there diminished in diameter. These spots, microscopically examined, showed a nearly complete disappearance of the ganglionic cells and nervous elements between them; a thickening, with nuclear proliferation, of the neighbouring blood-vessels; the intra-medullary portion of anterior roots, with but few, thin, axis-cylinders. Similar,



though much less marked changes existed throughout both anterior tracts of grey matter. The posterior portions of the cord and antero-lateral columns presented nothing abnormal, but an increased size of some axis-cylinders at one or two points. In the sciatic nerves some bundles of fibres were found converted into tracts of connective tissue.

The usual degenerative changes were observed in the paralysed muscles. Respecting the *pathogeny* of the disease, Dr. Müller points out that, owing to the physiological and histological peculiarities of the anterior horns, as well as to the marked independence of their capillary plexus, the possibility of their being seized upon by a pathological process, with but little implication of the antero-lateral and posterior tracts, is at once made evident. Then, again, the symptoms, both negative and positive, agree so well with what is known of the localisation of functions in the cord, that we feel justified in calling the disease a poliomyelitis. Lastly, such a case as Schultze's, where the whole length of both anterior horns was shown to have been the seat of inflammatory phenomena, with next to no lesion of the rest of the cord, confirms our present theory of the disease.

The *etiology* is yet obscure. With regard to age, the third septennium seems to be the most favourable for the development of the disease; the age of 16 out of 47 patients ranges between 14 and 22. Out of 40 cases, 19 occurred in August-September; January shows 7 cases, the remainder being distributed over the other months. As to sex, males form nearly two-thirds of the patients. As in many other diseases, exposure to cold seems to act as exciting cause in adult poliomyelitis. In 25 out of 47 cases this was emphatically the case.

The *diagnosis* of the typical disease is easy. Dr. Müller, however, fully discusses the differentiation of those cases where mistakes might be possible; but our space prevents our following him in the interesting details he gives. The *prognosis*, "quoad vitam," is favourable, and the course has been sufficiently made clear in discussing the electrical condition of the muscles. With regard to the *treatment*, the author is very partial to hypodermic injections of ergotin, with atropia during the hyperæmic stage. Afterwards a tonic regimen, with iodide

of potassium and wet-sheet packings, to stimulate the resorption. The paralytical symptoms have often been combated with strychnia. But we agree with the author in rejecting this drug as useless in poliomyelitis. The only chance we possess to hasten and extend the natural process of restoration is the rational and persistent use of electricity. The treatment must be begun very early, and carried on patiently for months, nay years, if necessary. The best plan is to apply the positive pole (using a *large* plate; the usual sponge-holders are worse than useless) to the cervical or lumbar enlargements, for about five minutes, whilst the negative is placed on the affected muscles. A few interruptions, or voltaic alternatives, are then to be made in order to bring about contractions. Voluntary exercise, and promoting the warmth of the limbs, especially by massage, are valuable auxiliaries.

We recommend Dr. Müller's monograph to the attention of our readers, and thank him for his solid contribution to the pathology of the spinal cord.

A. DE WATTEVILLE.

## Clinical Cases.

### FIVE CASES OF DISEASE OF THE BRAIN, STUDIED CHIEFLY WITH REFERENCE TO LOCALISA- TION.

BY CHARLES K. MILLS, M.D.

*Neurologist to the Philadelphia Hospital.*

- I. Softening of the Pons Varolii.
- II. Tumour of the Pons Varolii.
- III. Localised Tubercular Meningitis.
- IV. Localised Tubercular Meningitis; Internal Pachymeningitis.
- V. Multiple Cerebral Embolism.

THE cases contained in this paper are, I think, of value from a number of stand-points, but my remarks upon them will be brief, and will chiefly refer to their bearing upon the question of cerebral localisation. Much of pathological interest is taught by them in regard to such subjects as thrombosis and embolism, necrotic and inflammatory softening, meningeal tuberculosis and pachymeningitis, and primary and secondary disease of the cerebral vessels; but too elaborate a discussion of separate features would have extended the article to unwieldy limits. Even with reference to localisation, the cases are, as far as possible, allowed to speak for themselves. Cases of multiple or diffused lesions, such as three of the five here presented, are not, as some suppose, without value in the study of the problem of the cerebral functions. If, for instance, we have a symptom-picture, which points with great positiveness to lesions of the cortical motor zone, and if, after death, such lesions are found, we should not disregard them altogether because of complicating conditions. In every field of medicine we are compelled to resort to processes of

comparison and exclusion, in order to arrive at a satisfactory explanation of special manifestations. The all-important point is that we shall be honest and thorough in our investigations.

*CASE I.—Softening of the Pons Varolii—Right Hemiplegia followed by General Paralysis—Rapid Elevation of Temperature before Death.*

B —, aged 40, was admitted to the Philadelphia Hospital on May 20, 1879. He stated that three weeks before, while lying down, he suddenly felt as if his head was spinning round. He tried to get on his feet, but could not do so on account of the vertigo and a general loss of strength. He did not lose consciousness during the seizure. His speech became so "thick" that he could not be understood, and he found, on rallying from the first effects of the attack, that he was partially paralysed in the right arm and leg, and that his face was a little drawn to the left.

For several weeks previous to this seizure he had suffered with headache. He had abused alcohol, and also had a history of syphilis.

During the three weeks previous to his admission he had been slowly improving. He was hemiplegic on the right side, and was weak in all his limbs, but he could sit up and could walk around, holding on to the beds and chairs with his left hand. The paralysis was more decided in the right upper extremity than in the lower, but it was well marked in both. He had a slight left convergent strabismus, and his mouth was drawn a little to the left, but his face was not otherwise affected; no ptosis, external strabismus, lagophthalmus, or other symptoms, except those noted, being present. He could converse without difficulty, although his speech was not perfectly distinct, owing apparently to slight lingual and oral weakness. He had full control of rectum and bladder. Sensation was well preserved. He was pale and haggard.

The above was his condition during the first twenty-four hours after coming under observation on the 20th of May. On the 21st, he called the attention of the nurse to the fact that he had suddenly become paralysed in the left arm; during the same day his left leg was attacked; he became much weaker in every respect, and had to take to his bed. He did not change notably in his symptoms until the 23rd, when he began to complain of severe frontal headache. On this day his urine was examined, and was found to contain neither albumen, tube-casts, nor sugar.

On the 24th he had a peculiar seizure, which, as I chanced

to be present in the ward at the time, I was fortunate enough to witness. His face was pale and anxious, and he broke out into a profuse general perspiration. The left internal strabismus was observed to be much more marked than it was on admission. Respiration became difficult, but it was not stertorous; he breathed in a jerky manner, and was almost constantly making a puffing sound with his mouth, at which also he frothed a little. He could not speak, and could not thrust his tongue beyond his lips. On talking to him, he seemed to understand all that was said to him, and would make futile efforts to reply; he would become highly excited, looking at those around him with a most beseeching expression, while the tears suffused his cheeks, as if appreciating the helplessness and hopelessness of his situation. He could only open his mouth enough to separate his teeth from a quarter to half an inch. Liquid food and medicine could be taken, but he had some difficulty in swallowing, owing to a tendency to regurgitation. He was helpless in all his limbs, but the loss of power seemed still more prominent on the right. Slight flexions were present at the wrist, in the fingers of the right side, and at both knees. At intervals he had considerable convulsive tremor, affecting especially the right side, and the leg more than the arm. His pulse was 88 and weak; his temperature, taken in the rectum, was  $100\cdot4^{\circ}$  F. Involuntary passages did not occur; nausea and vomiting were not present. His eyes and neck, at this time, showed no tendency to deviate either to the right or left.

On the 25th, 26th, and 27th, he continued in nearly the same condition, having improved slightly, under counter-irritation to the back of the neck and extremities, and the use of stimulants. He still perspired freely nearly all the time; he remained anxious-looking and emotional, his breathing became a little better, but continued to be of the same puffing, jerky character; he did not regain the power of speaking, thrusting out his tongue, or swallowing without difficulty; the strabismus and helplessness of his limbs did not change. His pulse was weak, and ranged between 100 and 112. His temperature, taken in the rectum, varied but little during these three days. The record was as follows:—

	Morning.		Evening.
May 25	. . . $100^{\circ}$ F.	. . .	Not taken.
„ 26	. . . $100^{\circ}$ F.	. . .	$100\cdot4^{\circ}$ F.
„ 27	. . . $99\cdot8^{\circ}$ F.	. . .	$100\cdot3^{\circ}$ F.

On the morning of the 28th he became much worse. Sweating became more profuse; his paralytic symptoms deepened;

about midday involuntary discharges took place; his head and eyes, for the first time, showed a constant tendency to turn to the *right*; his breathing became more and more irregular and difficult. His temperature shot up rapidly; at 7 A.M. it was 101.3° F.; at 3 P.M., 105.5°; at 6½ P.M., 108°; after this it was not taken. He died at 11½ P.M.

An autopsy was held fourteen hours after death. The pia mater exhibited slight general cloudiness, with scattered patches of extreme redness. On examining the base the basilar artery was found to be enlarged to nearly twice its usual diameter, its walls being thickened and rigid, and rough on the outside. Some of the small vessels, which go down from it into the pons, could be seen to be obliterated; the basilar trunk itself, however, was quite free and of good calibre, and was readily opened with the scissors. The other main arteries, and their primary and secondary branches, showed wide-spread evidences of atheroma.

The pons varolii was the seat of an extensive and interesting lesion, the following description of which is partly from notes made at the time of the post-mortem examination, and partly from careful subsequent investigation of the specimen. This lesion was an irregular area of softening, the centre of which was just below the centre of the pons. Superficially, the softening was an inch in greatest length, which was from above downwards and to the left of the median line, and three-fourths of an inch in width at its widest part, which was near its lower boundary. Vertically, the softening extended about one-fourth of an inch higher to the left of the median line; while horizontally, and below the centre, it reached about one-eighth of an inch farther to the right than to the left. It presented four spots of excavation, the intervening spaces being filled up with softened and broken-down tissue; one of these was a comparatively large central space, the second was to the left and above, the third to the left and below, and the fourth to the right and below the centre. The entire surface-softening was included in an irregular triangle, formed by joining these outer points of depression or excavation. On subsequent close examination, the central region of the pons was found to be scooped out to the depth of half an inch, and it was also irregularly invaded in all directions by softened tissue; but its upper and lower fifths, and lateral bands of from one-fifth to one-sixth of its width, were unaffected. The cranial nerves, superficially, were not involved. The floor of the fourth ventricle presented a healthy appearance.

An extravasation as large as the palm of the hand was present in the cardiac end of the stomach. The aorta was

atheromatous. Both kidneys were fatty, the degeneration being more advanced in the right than in the left.

*Remarks.*—Summing up this case, the chief points are found to be as follows: headache; a vertiginous seizure, which left partial right hemiplegia; left convergent strabismus; defective articulation; paralysis of the left arm coming on suddenly three weeks after the first attack, but without any special head or general symptoms; a third seizure, accompanied by pallor, anxiety of expression, great emotionality, profuse perspiration, difficulty in breathing and swallowing; inability to speak, to thrust out the tongue, or open the mouth widely; paralysis of both arms and both legs, but still more pronounced on the right; convulsive tremor, most marked in right leg; weak and frequent pulse; slight elevation of rectal temperature for four days. On the day of death, deepening of all the symptoms just noted, and, in addition, paralysis of bladder and bowels, conjugate deviation to the right, and rapid elevation of temperature to 108° F. Some interesting negative facts are, absence of anæsthesia, of nausea and vomiting, of true convulsions, and of changes in the urine.

At first sight this case might be looked upon as contradictory of the views of those who hold to the decussation at the anterior pyramids of voluntary motor fibres, but I doubt whether it should be so regarded. Leaving out the question of some possible peculiarity in the decussation, in regard to which I have not yet had the opportunity to determine positively, we have in the history of the case, conjoined with the results of the autopsy, a satisfactory explanation of all the manifestations. The paralysis following the first seizure was of the face and limbs of the right side, and it continued until the day of death to be most decided on the right. The autopsy revealed an area of softening in the pons, occupying the centre and both sides of the median line, and this was evidently due to closure by a thrombotic process of some of the little nutrient arteries given off from the basilar. The different vessels probably became obstructed at intervals corresponding to the several successive seizures which the patient experienced. The softening in the centre and to the left may, in this way, have taken place earliest, an order of events which would best account for the right hemiplegia. The spots of excavation, spoken of in the notes of the autopsy, possibly represented the point at which the successive attacks of softening occurred. Paralysis of both sides of the body did occur before the death of the patient, as might be expected from a lesion of the kind and in the situation described.

Convergent strabismus, inability to articulate, to thrust out

the tongue or to open the mouth widely, and difficulty in swallowing and breathing, as the cranial nerves were not affected superficially, point to the probable involvement of the nuclei or deep tracks of the abducens, facial, hypoglossal, motor-trigeminal, glossopharyngeal, pneumogastric and spinal accessory. The escape of the posterior columns and the floor of the fourth ventricle are of interest in connection with the absence of anæsthesia, of nausea and vomiting, and of true convulsions.

The temperature observations made upon this case, although limited, are sufficient to be of some value in reference to the question of a heat-centre in the pons. Supposing the existence of such a centre, it is probable that the first foci of softening did not implicate it directly, but as the necrotic area enlarged, this centre became eventually involved.

CASE II.—*Tumour of the Pons Varolii—Convulsions, Vomiting, Rotatory Movements towards the Left—Final Hæmorrhage into the Medulla Oblongata.*

J—, 35 years of age, seven years before coming under observation, began occasionally to have headaches, and a year later became subject also to temporal and orbital neuralgias. The headaches and neuralgic attacks gradually grew worse and worse, both as regards frequency and severity. Five years after his first headaches he began to have "spells," in which he would be unconscious for a moment or two, and would have twitchings about the mouth and in the hands. These seizures returned at intervals of from one to two months for about a year, when they were supplanted by general convulsions. Sometimes he would have several convulsive paroxysms in the same day. Before the attacks and during these days of spasmodic storm he suffered with terrible headache and uncontrollable vomiting. Often at other times, without convulsions, he had spells of nausea and headache. Ophthalmoscopic examination showed atrophy of both optic nerves. His memory had failed, and he was usually in poor spirits. No positive paralysis was ever observed, although he was never closely examined for slight deficiencies in strength. His bowels were obstinately constipated, and he suffered nearly all the time from fæcal accumulations. He occasionally had "cramps" in the stomach and legs. He denied venereal disease of any kind, but his wife had had several stillborn children. The bromides, potassium-iodide, mercurials, and measures to relieve special symptoms, such as constipation, were employed, but without doing more than slight temporary good.



Six weeks before his death I saw him in one of a series of terrible convulsions. Although the convulsion was general, his right side seemed to be more affected than the left. Lying on his back, the spasms had the effect of lifting up the right side of his body and causing him to work over towards the left, as if trying to get on his face in this direction; but before getting altogether on his left side, he would fall backwards again, when the same curious lifting and rotary movement would be repeated. Frothy saliva escaped from his mouth; his face was much contorted, and was pale and livid by turns. The seizure lasted in all three minutes; when he relaxed he was bathed in perspiration and went off into a stertorous sleep. He died at the close of a series of similar convulsions, his death being preceded by general paralysis and involuntary evacuations.

The post-mortem examination, made twenty-three hours after death, revealed a round white tumour, a quarter of an inch in diameter, in the pia mater just to the left of the centre of the anterior surface of the pons varolii. The entire anterior central portion of the pons was softer and more doughy than usual. Just at the junction of the pons with the medulla oblongata was a recent hæmorrhage, which had spread downwards to about the middle of the latter. No other lesions of the brain were discovered, and this was the only organ examined.

Microscopic examination showed the tumour found in the pia mater to be a granulation tissue, probably of syphilitic origin. Nothing but mere detritus masses could be detected in the softened portion of the pons, and it could not therefore be determined positively whether this was a broken-down gumma.

*Remarks.*—The important symptoms in this case were headache, neuralgia in a portion of the trigeminal distribution; *petit mal*, supplanted after a time by fully formed convulsions; convulsions general, but the movements more marked on the right half of the body; during the convulsions, execution of a rotatory movement towards the left; failure of sight and optic atrophy; loss of memory and depression of spirits; frequent spells of nausea and uncontrollable vomiting at the time of the seizures; obstinate constipation, and occasional cramps or spasms in the stomach and legs. The above are points carefully studied and noted when the observations were made. The case, however, was under my care several years since, when I was not so fully impressed as I am now with the importance of investigating minutely. Sensation was not

closely studied, although I remember distinctly that on several occasions the patient complained of numbness of one side of the face. No definite paralysis of face, arms, or legs, of muscles or muscular groups, was present, although the man showed general and gradually progressing weakness of all the limbs. The trigeminal neuralgia, with probable anæsthesia, the peculiar epileptiform seizures, the local cramps or spasms, and the excessive vomiting, were symptoms which pointed to tumour of the pons—to a strongly irritative lesion in this region. The headache, optic atrophy and mental failure are the accompaniments of cerebral tumours in various locations. General weakness of all the limbs is the condition found in a number of cases of central lesion of the pons. Even supposing the central softened mass on the anterior surface of the pons to be a gummatous tumour, some of the fibres constituting the motor tracks on both sides were probably uninjured. The peculiar rotatory movements towards the left, recall the experiments in which Schiff divided the middle cerebellar peduncles at the side of the pons, the result being the performance by the animal of a rotatory movement towards the side of the lesion. The pial tumour was a little to the left of the median line, although the actual destruction of the pons was central. The fibres which go to form the middle cerebellar peduncle were, no doubt, affected by pressure and directly, the irritation being greatest to the left. The lesions were so situated as not to have involved directly the great sensory tracts. The hæmorrhage which spread over the medulla oblongata was doubtless the immediate cause of death; and this was probably brought about by the impairment of the vessels by disease of their walls and by proximity to the growth, and by the rise in blood-pressure during the terrible epileptiform paroxysms.

CASE III.—*Localised Tubercular Meningitis, with Cortical Softening—Involvement of the Centres for the Limbs and Face.*

M. H—, 30 years of age, could not give a very clear and full previous history; but stated that one year before coming under observation she had been ill for several weeks; that she had had high fever, with great pain in her limbs, particularly in the right arm and leg. During and after this attack she suffered almost constantly from headache, and occasionally from dizziness. She had also been troubled with cough. Four months before her admission to the hospital, while standing over a fire cooking, her right arm suddenly fell helpless

to her side. About ten days later, the right side of her face became partially paralysed; and still later, but exactly when she did not remember, her right leg became weak. Two months before admission she had an inflammatory affection of the right eye, from which she had just recovered. She had kept on her feet most of the time, but says that she was feverish and weak, and was much worried with pain in her head.

On examination I found that the right arm and right leg, which were about equally affected, were markedly weaker than the limbs of the left side; they were decidedly paretic, but presented no contractures, and only slight wasting. The lower part of the right side of the face was also paretic, as was shown by slight drooping of the nostril and corner of the mouth, and twisting to the left of the mouth. She could not draw up the right angle of the mouth when directed to do so, as she could the left. The tongue was not deflected, but its movements were performed in a weakly, tremulous manner. The right eyelid drooped slightly. No strabismus was present. The condition of the pupil could not be determined, on account of an opacity which covered the right cornea almost entirely, and was the result of the recent keratitis from which the patient had been suffering. Both eyelids could be opened and closed, and frontal furrows were present on both sides. Her manner of speaking was hesitating and unsteady, as if from weakness of the oral and lingual muscles. She was not aphasic, in the sense of having any partial or complete loss of speech. She did not drop letters, syllables, or words; but talked along, without stopping, in a drawling, uncertain, slipshod sort of way. Sensation was unimpaired. Hearing was good. Smell was defective, but not lost on either side. She could not, of course, see with the right eye, with its opaque cornea; the sight of the left was poor. Ophthalmoscopic examination, by Dr. E. O. Shakespeare, revealed a beginning neuroretinitis. Her mind acted torpidly, and, judging from the results of questioning her in regard to her past history, her memory was of the poorest kind. She had full control of her evacuations. She had no history of vomiting or convulsions. Her appetite was poor, and she was constipated. No examination of pulse or temperature was at this time made. She had probably had venereal disease, but I could not determine this certainly. Percussion and auscultation showed partial consolidation of the apices of both lungs.

She was ordered potassium iodide and cod-liver oil. Sinapisms were also applied to the neck, and a calomel purge was given occasionally. She improved a little in every respect;

but particularly as regards her headache, which almost entirely left her. After remaining under treatment for five weeks, during which time she was never confined to her bed, she asked for her discharge, and left to return to her family; but twelve days later she was again brought to the hospital, but now in a helpless and semi-conscious condition, from which she never rallied, dying one week after her re-admission. A few points were again noted. The loss of power in the right arm and leg had become a marked paralysis. The leg could be moved up and down over a space of a few inches, as she lay in bed. The paresis of the lower facial fibres was more positive; and both ptosis and rotation outwards of the eye were present and well marked. At intervals the right leg, and the arm and leg of the *left* side were the seat of twitching or convulsive tremor. She had fever, with paroxysms of sweating; she was delirious at times; her pulse was frequent and irregular.

The autopsy was held sixteen hours after death. The dura mater of the left hemisphere was slightly adherent in its inner surface at two points along the parietal margin of the fissure of Rolando, one at the median end of the fissure and the other just above its centre. On pulling the membrane away, which could be easily done, a mass of soft yellow exudation was found at each place of adhesion. The pia mater, both anterior and posterior to the fissure of Rolando, presented evidences of inflammatory action, being more or less opaque, streaked, and hyperæmic. On attempting to remove it, at several points its under-surface was found to be adherent to the convolutions, and to be the seat of what appeared to be tubercular masses, with purulent exudation, the cortex being superficially softened at some of the points. The peculiar character and localisation of the pathological process in the pia mater and cortex may be understood from the following description: (1) The pia mater was opaque, thickened slightly, and adherent at points over the paracentral lobule and the upper extremities of both ascending convolutions, the cortex being softened over a spot one-fourth of an inch in diameter in the ascending parietal convolution, at its upper limit. (2) Deposits, yellowish exudation, and a space of superficial softening, half an inch in diameter, were present about the centre of the ascending parietal convolution. (3) Two foci of cortical softening were found in the ascending convolutions, on each side of the lower fourth of Rolando's fissure, with pial changes at corresponding points.

The sides of the fissure of Rolando, at its middle third, were bound together by inflammatory products.

The pia mater of the interpeduncular space was very opaque,

much thickened, and of a gelatinous consistence; and the changed condition of the membrane, but with less opacity and thickening, continued into the left fissure of Sylvius, and upwards from the horizontal branch of this fissure along the convexity of the hemisphere in the general region of the distribution of the Sylvian artery. Greyish nodules were found here and there along the vessels, and a few purulent streaks and hyperæmic spots, besides the special areas along the fissure of Rolando already described. The pia around the optic chiasm, and particularly on its left side, was markedly thickened and changed in appearance. All the vessels, nerves, and bodies, at the base, from the pons to the chiasm, were more or less involved in an inflammatory process of the membrane. The microscopic appearances pointed to a tubercular inflammation of the pia mater, confined chiefly to the central portion of the base of the brain, and to the territory of the left middle cerebral artery. The tubercular process had been most extensive, and its effects most severe, at the points along the fissure of Rolando, and over the paracentral lobule, which I have specifically described. Some development of miliary tubercles had probably occurred at points in the cortex itself. Small vessels had certainly been obliterated. The pia mater over the pons and medulla, over both occipital lobes, and along the edges of the longitudinal fissure, was much more injected than it is usually found, but no tubercular appearances were discovered outside of the territories detailed. The ganglia, and all portions of the brain, were examined, but no other abnormal conditions were found. I was not able to secure an examination of any other organs.

Several specimens of the diseased pia mater, with blood-vessels, and the softened cortex, and the eyes with parts of the optic nerves attached, were submitted to Dr. E. O. Shakespeare for microscopical examination, and to him I owe the following valuable report, and also the opportunity of examining the mounted specimens:—

“The specimens of the membranes and cortex of the brain, and of some of the larger cerebral vessels, were found in good condition for microscopical examination. Thin sections were made from the softened cerebral convolutions, with its attached pial covering, were stained with carmine, and were temporarily mounted in oil of cloves. The larger vessels were treated in a similar manner; and transverse sections of them, when placed beneath the microscope, showed the walls of many to be diseased. The lesion was limited almost exclusively to the adventitia and surrounding connective tissue. In general, only the outer part of the muscular tunic was affected. The

tunica intima showed scarcely any appreciable alteration. The lesion located in the outer coat of the vessel was characterised by a dense accumulation of embryonal cells in the meshes of its reticulum. Seen in longitudinal section, the vessels often presented a fusiform enlargement from point to point, an alteration which was restricted to the outer coat, and which rarely ever affected the calibre of the vessels. It was almost exclusively the arteries which were thus diseased.

“The sections of the cerebral cortex were made vertical to the surface of the softened convolution. They showed a variable number of caseous nodules, some of which were situated in the cortical substance near the surface, others in the thickened and inflamed pia mater. Upon minute examination, these caseous nodules were found to be composed of numbers of smaller caseous foci, massed together and united by an embryonal tissue, which presented the following peculiarities: in the portions at some distance from the minute caseous foci, embryonal cells were crowded together so thickly that it was impossible to discover any trace of the original structure. Scattered here and there in this embryonal mass were to be seen a number of what many authors figure and describe as ‘giant cells.’ On first view, and under a moderate magnification, these large multinucleated bodies certainly presented many of the characteristics of myélopaxes; indeed, at the very edge of the minute caseous foci, where the embryonal tissue was on the border-land of caseous degeneration, it was altogether impossible from an examination of these bodies at this location alone to say that they were not genuine ‘giant cells.’ Recurring, however, to the more remote portions of the embryonal mass, where the cellular tissue was not nearly so much altered, by a very careful study, under a very high power given by an excellent lens, there was no difficulty in recognising peculiarities in these large multinuclear bodies, which demonstrated their nature and construction to be different from those of true ‘giant cells.’ In the loose altered spots of the embryonal mass could very readily be recognised the lumen, wall, and peri-vascular lymph-space of small arteriole and other blood-vessels. Many of these vessels presented an unobstructed lumen; and yet the walls of all of them were altered. The lesion of the patulous vessels was again mainly limited to the external coat, which, together with the outer layers of the middle coat, was represented by a mass of embryonal cells, constituting a cylinder. The endothelia of the tunica intima were swollen, and presented enlarged nuclei, but did not appear to be proliferating. Sometimes the peri-vascular lymph-space would be filled more or less completely with large

embryonal cells, but it was usually almost empty. The swollen vessels presented corresponding alterations. In some less altered spots, many of the vessels, in the same condition above described as to their walls, were plugged with a granular, fibrinous clot, which entirely filled the lumen, and which enclosed some white blood-corpuscles, a few detached swollen endothelia, and sometimes small numbers of visible red blood disks. In these cases a granular fibrinous coagulation more or less completely obstructed the perivascular lymph space also. In the intermediate region, between the above-mentioned altered portions of the embryonal mass and a narrow zone immediately surrounding the minute caseous foci, were many multinuclear bodies, in which the outlines of the vessels could still be faintly recognised by the arrangement of the nuclei. In this region were some also, in which, without the aid of a previous study, no suspicion of a similar origin and mode of formation could be suggested by their appearance, every semblance of a transverse cut of a vessel having been entirely lost; and yet, throughout the different portions of the embryonal mass, multinuclear bodies were so numerous, and of such varied appearance, that every gradation could be traced between the sections of undoubted blood-vessels, the external parts of which were in a state of proliferation, and the multinuclear bodies in the neighbourhood of a caseous focus, which gave no hint of such an origin.

"In the brain substance, and in the pia mater at the border of the embryonal masses which surrounded the caseous foci, the arterioles and the capillaries were altered in the following manner. Their external portions showed a cellular irritation throughout their entire length; at points the irritation had gone so far as to result in a nodular, or fusiform, or cylindrical thickening of the outer wall of the vessel. In the immediate vicinity of these altered vessels the tissue was in a state of inflammatory hyperplasia or inflammatory softening.

"From the foregoing study of the microscopical characters of the various lesions, it must be concluded that the specific morbid process in this case was that of tuberculosis.

"Longitudinal sections of one optic nerve, at its entrance into the eye, were made and prepared for study. Examination of these showed the nerve to be in the early stages of a descending optic neuritis."

This report speaks for itself, showing clearly the existence of tuberculosis explaining satisfactorily the occurrence of the softening, and containing several points of special interest, such, for instance, as those relating to supposed "giant cells."

*Remarks.*—Although the autopsy revealed a somewhat diffuse meningeal inflammation, the only definite destructive lesions were those of the convolutions surrounding the fissure of Rolando. The inflammation, deposit, exudation and destruction of the cortex were greatest at the middle region of the left ascending convolution, where are situated the motor centres for the movements of the upper extremity. Motor paralysis of the right arm was the most marked of the paralytic manifestations. To explain the paralysis of the leg, we have thickening and adhesions of the pia mater over the paracentral lobule, with a small area of softening high up in the ascending parietal convolution. The nasal, oral, and lingual paresis have their explanation in the foci of softening and the tubercular granulations low down in the ascending convolutions. The lesions of the cortex were carefully and closely studied, a pencil-sketch, with measurements, indicating their intensity and exact location, being made at the time of autopsy, under my directions, by one of my resident physicians, Dr. Bell. Her disturbance of speech was an oro-lingual paresis rather than a true aphasia. The ptosis and late external strabismus, were probably due to the involvement of the oculo-motor nerve in the meningeal inflammation at the base. Both ophthalmoscopic examination and the microscope showed the association of optic neuritis with the meningitis.

CASE IV.—*Localised Tubercular Meningitis with Cortical Softening—Involvement of Oro-Lingual Centres—Internal Pachymeningitis of the Opposite Hemisphere.*

Y—, aged 65, was sent from the Blind Ward to the ward for nervous diseases, because he had had a slight apoplectic seizure. I could only learn in regard to him that he had been weak and thin for a long time; that he had been troubled with cough and diarrhœa; and that he had complained frequently of headache and giddiness. His mind acted very feebly; he seemed to understand what was said to him, but he was somewhat obtuse and slow in answering. His speech was peculiar, it might be described as slightly “staggering:” a little tremor was present in the tongue and the muscles about the mouth; he sometimes dropped a letter or syllable. He was drowsy, dosing the larger part of his time. The only evidence of loss of power in the face, which was carefully studied, was about the mouth, which was drawn slightly to the left, and the right angle of which drooped slightly. His tongue did not deflect to either side, but its movements showed weakness and some want of control. The uvula and soft



palate were not paralysed. Neither ptosis nor strabismus was present. Cataract operations had been performed on both eyes, and he was nearly blind, having only quantitative vision. His limbs were all very weak, but no distinct motor paralysis, more marked in one than in the other, could be made out, except that the grip of the right hand was poorer than that of the left. His right leg, however, had been amputated a few inches below the knee, about five years before coming under observation, which prevented a full determination as to paralysis on this side. Physical examination of both lungs showed that wide-spread and advanced tuberculous disease was probably present. He was troubled with an obstinate diarrhoea, and died, in ten days after admission, of exhaustion, which was largely due to this diarrhoea.

An autopsy was held twelve hours after death. The skull was normal. The brain weighed forty-three ounces. The inner surface of the dura mater of the convex portion of the right hemisphere, from the middle of the parietal lobe forward to the anterior extremity of the brain, was covered with a layer, from half a line to a line in thickness, of a red, yellowish-red, and reddish-brown colour at different points. This stratum could be readily scraped from the dura, leaving this membrane intact beneath. The same formation, but of less thickness, extended down the right side of the falx. The pia mater, beneath the changed dura, presented a slightly opaque, bluish-white appearance. After stripping up this membrane, it was found that all the convolutions of the convex surface of the hemisphere, within the area covered by the diseased dura, were much flattened, and of a reddish-brown or light rusty colour.

The dura mater of the left hemisphere was unchanged. The pia mater of this side was hyperæmic, and here and there through it were seen peculiar-looking straight streaks of red. On removing the membrane from the substance of the brain it was adherent at several points, particularly along the fissure of Rolando. Miliary tubercles were made out at scattered points over the postero-frontal and parietal lobes.

On the surface of the left hemisphere were four foci of superficial softening, corresponding to points where the pia mater was more or less adherent to the cortex, and presented on its under-surface spots of yellowish exudation. The softening was only from one to two lines in depth, and was of a whitish-yellow colour; it had probably resulted from the obliteration of small vessels by the tuberculous process, which was more intense in the pia mater at these positions than elsewhere. These softened areas were situated as follows: (1) An

irregular area, five-eighths of an inch wide at its upper boundary, and narrowing below to one-fourth of an inch, at the level of the middle third of the fissure of Rolando, its posterior margin being eleven-sixteenths of an inch in front of this fissure; it included small portions of the second, third, and ascending frontal convolutions. (2) A spot, about the size of a half-dime, in the ascending parietal convolution, about the junction of the middle and lower thirds of the fissure of Rolando. (3) A spot, two-thirds of an inch vertically, and one-fifth of an inch wide, extending on both sides of and one-third of an inch below the lower extremity of the same fissure. (4) An area, five-eighths of an inch in width, and extending across the second temporal convolution, an inch backwards from the Sylvian fossa.

A very slight depression was found about the centre of the ventricular surface of the left corpus striatum.

Both lungs were infiltrated with miliary tubercles, the upper lobes being most affected. Cheesy metamorphosis had taken place in several localities, and an abscess had formed in the right apex. The cardiac valves were all-sufficient, and nothing abnormal was found in the examination of the heart except a single calcareous plate on the outer side of one of the aortic crescents. The right kidney contained a brown encysted stone, and two similar stones were found in the substance of the left kidney. Both small and large intestines presented a remarkable condition of ulceration; fifty-two distinct ulcerative foci being counted in the former, and twenty in the latter.

Microscopical examination confirmed the macroscopic evidences of the existence of tuberculosis of the pia mater.

*Remarks.*—In this case we have a tuberculosis of the pia mater of the convexity of the left hemisphere. The tubercles discovered were fewer in number, and the signs of inflammation were less marked than in the last case; speaking generally, the same portion of the convexity was attacked in both, but in the present case the base escaped. Limited tuberculosis of the pia mater is not so rare as many neurologists suppose.

In regard to localisation, it will be seen from a study of the areas of softened cortex that portions of the arm, face, and speech-centres were implicated. The softening was very superficial, and the paresis of the right upper extremity may or may not have been the result of the lesions; but I am inclined to attribute the peculiar speech and the paresis of mouth and tongue to the cortical destruction, as the oro-lingual centres of Ferrier were so distinctly involved. In regard to the softening of the second temporal convolution, I have nothing to say in

connection with the symptomatology of the case. The special senses could not be studied to any purpose.

The internal hæmorrhagic pachymeningitis of the right hemisphere was in itself a most interesting lesion, but a lengthy discussion of it is foreign to my purpose in the present paper. Although the case was one complicated by many conditions, some of the patient's symptoms were doubtless due to the cerebral compression, which the autopsy showed had been exerted by the chronic pachymeningeal disease. Drowsiness was one of these symptoms; and the headache, vertigo, and weakness of the left half of the body may also have been partly, at least, due to the dural affection. Pachymeningitis has not unfrequently been observed in cases of tuberculosis of the pia mater, lungs, and other parts of the body, and also in connection with atheroma and affections of the kidneys.

*CASE V.—Multiple Cerebral Embolism—Softening of the Corpus Striatum and Internal Capsule, and widely distributed Cortical Softening—Hemiplegia with Aphasia.*

C—, aged 45 years, when she came under my care, was not in a condition to give any information herself, and the few facts in regard to her previous history were obtained from her relatives and friends. During three years she had suffered with pains in her limbs. She was brought to the hospital on the 18th of July. Late in the preceding winter she had a stroke, which paralysed the right half of the body and affected her speech. Afterwards she never used more than three expressions, namely, "Yes," "No," and "Do you know?" She became irritable and emotional. On the 6th of July she had a second apoplectic seizure while lying down. She could not move from the position she was in, but was not unconscious. From this time she was helpless, was unable to feed herself, had involuntary passages, and had some fever with delirium at night. She often carried her left hand to the head, as if it was the seat of pain.

She appeared to understand much that was said to her when she was first seen, but she could not answer questions correctly. Her vocabulary now consisted only of the two words "Yes" and "No," and she used one of these for the other indifferently. The mouth showed a scarcely perceptible twist towards the left, so slight that I could not feel sure that it was not a deviation such as is sometimes seen in those in health. She could not get her tongue beyond her lips. She could swallow, but with some difficulty. She could open and

shut both eyes, and no noticeable paresis was present in the upper part of the face. The right upper extremity was powerless, and was wasted from the shoulder to the finger-ends; the fingers were slightly clawed. The right leg was also helpless, but not so absolutely as the arm; the limb was wasted, the thigh much more than the leg below the knee. She had no appetite; she did not seem to care for food at all. Sensation, roughly tested, seemed to be retained; she shrank from the pricking of the æsthesiometer, but this may have been partly a reflex manifestation. Patellar reflex on the right side was decidedly exaggerated, and a slight clonus could be developed from the right ankle. The above notes on her condition were made July 20th. She lived until the 30th, gradually growing worse, although she sometimes rallied a little for a few hours. She had more or less fever all the time, her night delirium continued, her pulse became frequent and irregular, and profuse sweating often occurred. Her temperature was taken both morning and evening, and in both axillæ, from July 24th to the 29th. The temperatures registered in the right axilla, the side paralysed, were higher than those in the left. The evening temperatures were usually considerably higher than those of the morning. The averages of the observations were as follows:—

	Right Axilla.		Left Axilla.	
Morning	.	99·7° F.	.	99° F.
Evening	.	101°	.	100°·2.

A post-mortem examination was made twelve hours after death. The dura mater was paler than usual, and the pia mater was everywhere so anæmic and transparent that the convolutions could be readily traced before its removal. The brain weighed forty-one ounces. No less than seven areas of true cortical softening were found, as follows: (1) A spot, about two-thirds of an inch in diameter, on the left side, where the first frontal, second frontal, and ascending frontal convolutions come together. (2) One, a third of an inch in diameter, in the left third frontal convolution at the bottom of a secondary fissure. (3) A similar focus on the outer edge of the left island of Reil. (4) An irregular area, two-thirds of an inch in its greatest length, about the middle of the first temporal convolution of the right side. (5) A large foyer, which began an inch to the right of the longitudinal fissure, and just anterior to the transverse occipital fissure of Ecker, and extended backwards, expanding like a fan to the posterior limit of the upper surface of the right occipital lobe. (6) A similar foyer in the left occipital lobe, which began at a corresponding point, and spread in the same general direction, but only for about half

the distance of the softening on the right side. (7) A small spot, one-third of an inch in diameter, on the right posterior aspect of the cerebellum.

On cutting into the left lateral ventricle, a small yellow spot was visible on the surface of the corpus striatum at the beginning of its cue or tail. On incising this, the ganglion beneath was found to be softened and partly diffuent, the destruction being deep enough to involve a portion of the internal capsule. No other lesions of convolutions, tracts, or ganglia were discovered, after diligent search. The blood-vessels, particularly those of medium and small calibre, showed at intervals evidences of inflammatory changes in their walls. I succeeded in finding emboli lodged in vessels leading directly into the softened occipital areas. At a number of points in the branches of the middle and posterior cerebrals, at more on the left side, were seen what appeared to be plugs, or minute blood-clots, some loosely and some firmly lodged.

Pleuritic adhesions, which were particularly firm and dense on the posterior aspect of the left lung, were found on both sides. The lungs were œdematous, but contained neither deposits nor cavities. The heart was dilated, and its walls were in a condition of fatty degeneration. No valvular lesions, however, were present. Both kidneys were atrophied, granular, and tough.

The vessels were carefully stripped from the brain, and these, with specimens from the softened areas, were examined microscopically by Dr. E. O. Shakespeare, who reported as follows: "The walls of the vessels were in a state of cellular hyperplasia. The endothelial cells covering the trabeculæ of the finer fibrous network of the pia mater were swollen, granular, and sometimes contained more than one very distinct nucleus. In the meshes of the network in many places could be seen a coagulum of granular fibrine, enclosing white blood corpuscles, a few red ones, and some detached and swollen endothelia. In a few of the largest vessels an embolus could be distinctly recognised; and at the location of the plug, the inner, middle, and exterior coats of the vessels were much irritated and sometimes inflamed. The diseased cerebral substance presented at different points the usual appearances of inflammatory and necrotic softening. The pia mater attached to the softened cerebral tissue showed inflammatory thickening, and the appearances commonly present in subacute meningitis."

*Remarks.*—How shall we associate the symptoms presented by this case during life with the numerous lesions found after death? Such a multiplicity of diseased areas might at first

tend to throw us into confusion. The major portion of the dissociated hemiplegia was probably due to the softening of the left corpus striatum and adjacent internal capsule; the aphasia to this lesion, or conjointly to it, and to those of the third left frontal convolution and the island of Reil. The softening (1), at the junction of the first, second, and ascending frontal convolution, included the anterior upper portion of the general region for the superior extremity. It will be remembered that in the history given the paralysis and wasting were stated to be much more marked in the upper than in the lower limb. This fact was carefully determined, measurements of the limbs having been made both before and after death. The greater completeness of the brachial paralysis and wasting may have been attributable to the partial involvement of the general arm centre in one of the lesions, both the voluntary-motor centres in the cortex, and the sub-voluntary or automatic centres in the striate body probably being destroyed. The cortical areas of softening were not high enough to implicate the supposed centres for the leg; and the paralysis and atrophy of this member must, I think, be attributed alone to the lesion of the caudate nucleus and adjoining tracts. The wasting of the right thigh out of proportion to that below the knee, which strongly attracted my attention, is an observation not to be disregarded, although I have no special explanation to offer. I am satisfied that, with all the advances that have been made in this direction, we do not study our cases minutely enough. Many facts of value may yet be learned from the commonest examples of hemiplegia, by a closer study of symptomatology. Differences in the amount of wasting in various parts of the same limb, the greater preponderance of paralysis or spasm in certain muscles or muscular groups, and many other similar points, need to be more thoroughly investigated with reference to the effect and position of the lesions found post-mortem. The exact limitations, superficial and deep, of lesions of the basal ganglia, should be determined. In this case the head of the striate body escaped, but the softening extended below the caudate nucleus. Decided facial paralysis was absent, although a very slight paresis of the lower right facial region seemed to be present. The cortical facial zone, as usually given, just escaped. The tongue and the muscles concerned in swallowing were undoubtedly affected. Owing to the condition of the patient, hearing and the special senses generally could not be thoroughly investigated, so that I am not able to say whether any defect of hearing resulted from the temporo-sphenoidal lesion. In regard to the large foyers of softening in both occipital lobes, and the cerebellar

focus, if any inferences in regard to the lesions in other regions have been correct, the effects produced by these were latent, or were not of a positive character. Ferrier regards the occipital lobes as specially related to the visceral or organic sensibilities, but is doubtful whether I would be justified in referring such symptoms as impaired appetite and involuntary discharges, which occur in so many conditions, to any localised cerebral lesion. Sensation, so far as it could be tested, seemed to be unimpaired.

It may be worth while to give a passing notice to such symptoms as patella reflex, temperature, and fever and delirium. Very commonly, as in this case, in hemiplegics and monoplegics, tendon reflex is exaggerated on the paralysed side, owing probably, in part, at least, to the fact that the parts below the lesion are more or less cut off, or from the restraining influence of the higher brain. The temperature was slightly elevated, as in many instances of localised cerebral disease; it was also higher on the paralysed side than on the other. The case being one of demonstrable multiple embolism of vessels of appreciable size, Bastian's hypothesis in regard to fever and delirium being due to capillary occlusions becomes worthy of consideration. The conditions were such as to bring about such occlusions.

Pathologically, this case is a most interesting illustration of multiple cerebral embolism. It is probable that on several occasions particles from a partially-organised blood-clot were carried into the circulation, and lodged in the cerebral vessels. Some of these became permanent fixtures at their points of lodgment; others were immediately swept out again. Necrotic softening took place in some instances in the districts supplied by the occluded vessels; in others this process was probably prevented by collateral circulation. As has been noted, plugs were discovered in a number of places in branches of the middle and posterior cerebral arteries, these in some cases apparently not having caused necrobiosis. In some recent experiments made in conjunction with Dr. A. J. Parker, of Philadelphia, we found the communications between the three chief vascular territories of the brain freer even than is held by Heubner, and far freer than Duret would have us believe. Our inspections so far have been made only on two brains, and great individual differences may and probably do exist; but the fact that in these two sets of experiments we found communicating vessels at least two millimetres in diameter, shows that parts of the brain may frequently be saved from softening by anastomosing vessels. This is contrary to the views advanced by me in a Review of the subject of *Cerebral Localisation*

(‘American Journal of the Medical Sciences,’ July 1879), in which I state that up to the time of writing, my clinico-pathological observations were corroborative of the views of Duret, Cohnheim, and Charcot. Subsequent pathological experience, and the experiments just referred to, have, however, led me to a somewhat different conclusion, although I do not yet feel satisfied in regard to the question. Multiple embolism is probably more frequent than is usually supposed, occluded vessels which do *not* occasion softening often escaping attention.

The inflammation of the pia mater was confined to that portion of the membrane in immediate relation with the necrotic districts.

The inflammation of the walls of the vessels, which was so marked, may have been partly aroused by the irritation produced by the numerous embolic foci, partly it was due to these vessels coming from the zone of inflammation around the softened areas.

The co-existence of fatty degeneration and dilatation of the heart with contracted kidneys affords a plausible explanation of the formation of the supposed clot. A tendency to the production of cardiac and other thrombi is well known to exist in diseases such as fatty heart, which cause marked impairment of the power of this organ.

The areas of softening observed were more numerous in the left than on the right half of the brain, which is in accordance with the well-established and easily explained fact that emboli are much more frequently carried into the left carotid than into the right. The occurrence of bilateral softening of the occipital lobes is deserving of passing attention. A tendency to bilateral embolism has been noted by several observers.



## CASES OF INTRACRANIAL TUMOUR.

BY JAMES ROSS, M.D., MANCHESTER.

CASE 1.—JOHN THOMAS GOULD, æt. 14 years, was admitted into the Royal Infirmary on March 5, 1877, under the care of Dr. W. Roberts, to whose kindness I am indebted for permission to publish the case. He was a strong and healthy boy until a few months ago; his parents were also healthy, and there was no family history of consumption or of any other constitutional disease. He was a brick-setter by trade, and three months previous to admission fell from a ladder and struck the back of his head on the pavement, and since that time he has suffered from more or less constant occipital headache.

*Condition on admission.*—As he lies in bed he can move his legs freely in any direction; but on attempting to walk, the feet are alternately projected forwards, and the heel comes down with a thump, producing a gait similar to that of locomotor ataxy. He cannot maintain the erect posture unsupported; and when all external aid is momentarily withdrawn, his head shoots downwards and forwards, as if the body were about to revolve round a transverse horizontal axis. When the patient is caught in the act of falling and raised again to the erect posture, he complains of dizziness, and sees objects revolving from right to left. On directing his eyes to the right, a slight oscillatory movement of the eyeballs is observed, but there is no nystagmus when he looks straight before him. The patient is almost quite blind in the left eye; but he can distinguish objects clearly with the right eye.

When the right eye is fixed on an object, such as a finger twelve inches in front, a second finger moved laterally and to the right is seen until it is almost nine inches from the first, showing that the field of vision to the right is not sensibly diminished. But starting again from the finger on which the eye is fixed, and moving the second laterally to the left, the

latter disappears from view when it is from one to two inches removed from the former. I say from one to two inches, because his replies are not always the same, thus indicating that the sensitive and blind portions of the retina are not separated from one another by a sharp and defined border, but fade insensibly into one another. The pupils are dilated, equal, and respond slowly to the stimulus of light. An ophthalmoscopic examination reveals double optic neuritis with swollen disc; but there are no patches of exudation or atrophy. The other special senses and the mental faculties are unaffected, the urine is free from sugar and albumen, the appetite is good, and all the other functions of life are normal. He was ordered iod. potass., grs. x., to be taken three times a-day.

*March 31st, four weeks after admission.*—It is evident that the symptoms have altered considerably. The patient now habitually lies either on his back, or slightly inclined to one side. He cannot sit erect without support, but he has still some degree of voluntary control over his legs, although the movements are feeble. Cutaneous sensibility is impaired in the lower extremities; he can still feel when touched, but he cannot localise the touch well. The sensibility to variations of temperature and to pain is also impaired. There is complete blindness of both eyes, but there is only slight atrophy of the optic discs. Two days ago he passed his urine in bed for the first time. His bowels are very constipated, and there is general emaciation.

*April 15th.*—He complains much of frontal headache. There is complete anaesthesia, and entire loss of voluntary motion of the lower extremities; the stools and urine pass involuntarily, and there is a large bed-sore over the sacrum. He cannot hear the ticking of a watch so well with the left as with the right ear.

*May 14th.*—There is complete anaesthesia of all parts below a line passing round the body on a level with the anterior superior processes of the ilium. Reflex irritability is entirely abolished in the lower extremities, as tested by tickling, pricking, and the faradic current. The muscles of the legs and thighs do not respond in the slightest to the faradic current. The calf of the right leg measures  $9\frac{1}{4}$  and that of the left only  $7\frac{1}{4}$  inches. Each thigh measures  $9\frac{3}{4}$  inches. The muscles of the thighs and of the right leg appear only to be emaciated in proportion to the rest of the body; but it is manifest that the muscles of the left leg are specially atrophied. There are deep bed-sores over the sacrum, the prominences of both thighs, the external malleoli of both

ankles and the inside of the left knee. The intelligence is greatly blunted, and he lies in a half stupor, but replies readily to any simple question asked him. His pulse is very feeble, and beats about 140 in a minute; but he still eats any food which the nurse brings him. About a week ago it was noticed that the left eyelid was only half closed when he was asleep, and he could not close it entirely by a voluntary effort. There was also slight paralysis of the left facial muscles, so slight that the difference between the two sides could scarcely be detected when the face was quiescent, but was recognisable when the patient smiled. This affection of the facial nerve only lasted a few days, and has now entirely disappeared. The state of his hearing cannot be satisfactorily tested, owing to his apathetic condition.

From this time he lingered on without any further special symptom manifesting itself. He became more and more apathetic and extremely emaciated, and died on July 3rd, four months after entering the Infirmary, and seven months after the fall, to which in all probability the origin of the disease is to be traced.

*Section cadaveris, twenty-four hours after death.*—The body is greatly emaciated. The calf of each leg measures 7 inches, and each thigh  $7\frac{1}{4}$  inches. The sacrum, both trochanters, and the external malleolus of the left foot are exposed and denuded in consequence of extensive bed-sores. The tips of both ears are also ulcerated, as well as the inside of the left knee.

On removing the calvarium the brain appears to project, and the convolutions are flattened. The sinuses and the veins on the surface of the brain are gorged with blood. The substance of the brain is somewhat soft, and the ventricles are distended with fluid; but the cerebrum is in other respects healthy. Some grumous turbid fluid escaped from between the cerebellum and corpora quadrigemina during removal of the brain. On inspecting the cerebellum the edge of a tumour is noticed between the anterior surface of the middle lobe at its inferior margin and the posterior surface of the medulla oblongata; while the superior end of the tumour can be seen between the cerebellum and corpora quadrigemina. On making a vertical section of the cerebellum in the middle line down to the floor of the fourth ventricle, the tumour is seen to occupy the whole of the anterior part of the middle lobe of the cerebellum, being somewhat more developed on the right than on the left side. In consistence the tumour is soft, brain-like, of a greyish-red colour, the centre being so softened as to have left a small cavity from which the turbid fluid already mentioned had escaped. The tumour is not circumscribed;

its margins gradually blending with the surrounding nervous tissue. The growth passes along the right superior peduncle of the cerebellum to reach the corpora quadrigemina, and the latter are somewhat softened and flattened. On opening the spinal canal the cord is seen to occupy the whole of the cavity transversely, the diameter of the cord being about one-third larger than that of a healthy cord. The whole of the cord feels brawny, like bacon. On making transverse sections from above downwards, half an inch apart, the membranes (the cortical layer of the neuroglia?) in the cervical region are seen to be slightly thickened, and the cord is softened; but in the upper dorsal region the spinal pia mater, especially on the posterior aspect, is three times as thick as in a healthy cord. In the middle of the dorsal region the new growth forms a dense well-defined tumour, which pressed on the cord from behind forwards, so that only a small part of the anterior segment of the cord is left. In the lumbar region, again, the tumour surrounds the cord, so that a central core, about the size of a goose quill, of softened nervous tissue is all that is left to represent the spinal cord. The substance of the tumour is of the colour and texture of bacon, and much denser than the tumour of the cerebellum.

The left lung is adherent to the chest walls. Both lungs are healthy. The heart is normal. The abdominal organs are healthy. Subsequent microscopic examination showed that the tumour consisted of small delicate cells imbedded in a finely granular substance. The growth in the cord presented similar microscopic characters to the cerebellar tumour; but contained a larger amount of intercellular substance.

*Remarks.*—The first time I examined the patient, my diagnosis was “tumour, probably a glioma, situated in the inferior portion of the middle lobe of the cerebellum, and pressing forwards on the corpora quadrigemina.” The reasons for regarding the case as one of intracranial tumour are so manifest as scarcely to require mention. They are: the history of an injury to the head, the gradual development and progressive character of the symptoms, the constant headache, and the existence of double optic neuritis. My reason for believing that the tumour was situated in the inferior portion of the middle lobe of the cerebellum was the remarkable manner in which the head and shoulders shot forwards and downwards, as if the patient were about to revolve round a horizontal axis. The circumstances in favour of the tumour being a glioma were these: if the diagnosis with respect to the localisation was correct, the tumour probably grew in the substance of

the nervous tissue itself, and not from the membranes; the appearance of the patient and the family history were against tubercle; there was no evidence of congenital syphilis; and the age of the patient put cancer almost out of the question.

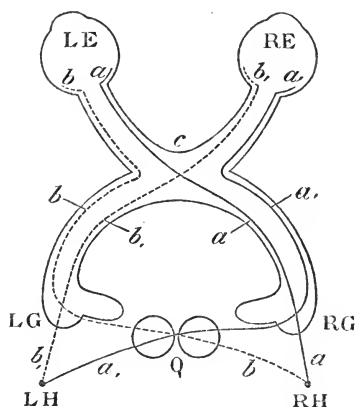
My reasons for thinking that the tumour pressed forwards upon the corpora quadrigemina were that I thought this would to some extent explain the excessive staggering present during attempts at walking; but much more that it would explain the great impairment of vision present at such an early stage of the disease. But the peculiar character of the disorder of vision demands an explanation; and for this purpose let us take Charcot's scheme of the decussation of the optic tracts as our guide.

In this case, vision was almost totally lost in the left eye, and the state of vision of the right eye simulated nasal hemiopia. I say simulated, because it was not a case of hemiopia at all. The condition of vision in both eyes was that of amblyopia in its progress towards amaurosis. The amaurotic condition was nearly reached in both sides of the retina in the left eye; but in the right eye the right half of the retina had become amaurotic, while vision was tolerably good in the left half; and between the two halves of the right retina there was a relatively broad border land, where the comparatively good vision of one side faded gradually into the blindness of the other. Such a condition as this could not, therefore, have been caused by the pressure of a tumour on the commissure or optic tracts; it was not likely to have arisen directly from the optic neuritis, inasmuch as there was no atrophy of, or patches of exudation on the disc; it must therefore have been caused by a lesion interfering with the optic fibres, either directly or indirectly, beyond the termination of the optic tracts in the external geniculate bodies.

It appeared to me very probable that a tumour pressing on the right side of the corpora quadrigemina and extending gradually to the left, would produce the condition of vision met with in this case. The whirling of objects from right to left which the patient described, showed that there was a greater amount of irritation of the right than of the left lobe of the cerebellum, and this rendered it probable that the tumour of the middle lobe extended farther to the right than to the left.

Suppose, then, that a tumour is pressing on the corpora quadrigemina from behind forwards, and from right to left, the fibres (*b a*) coming from the left eye, and meeting at RH, would be first interfered with, then the fibres (*a'*) coming from the right half of the retina of the right eye, would be intercepted

in their passage behind the corpora geniculata to the opposite side. The fibres *b'* coming from the left half of the retina of the right eye would be the last to be injured, so that the condition of vision which was present in this case would be produced. It may be urged that Charcot's scheme of decussation is merely diagrammatic, and that the points *LH* and *RH* are supposed to represent positions in the cortex of the hemispheres; but my reply is that I am only making a diagrammatic use of it. If there is a semi-decussation of the optic nerves in the chiasma, and if the fibres which do not cross in that place decussate behind the corpora geniculata, then, whatever may be the further course of these fibres, some such effect as that



*L E*, and *R E*, left and right eyes; *c*, chiasma; *L G*, and *R G*, left and right geniculate bodies; *Q*, corpora quadrigemina; *L H*, and *R H*, left and right centres of vision; *b* and *a* nerve-fibres from left and right sides respectively of left eye; *b'* and *a'*, corresponding fibres from right eye.

indicated would be produced by a tumour pressing from behind forwards, and from right to left on the corpora quadrigemina. This at least was the process of reasoning by which I came to the conclusion that the case was one of tumour of the anterior part of the middle lobe of the cerebellum, inclining to the right side, and pressing forwards on the corpora quadrigemina, and this conclusion was verified to a considerable extent by the post-mortem. One serious objection I always felt to this view was, that it was not manifest how the floor of the fourth ventricle could escape under such circumstances; and yet there was no sugar in the urine, no polyuria, and the breathing was uninterfered with. The autopsy explained this. It showed that the corpora quadrigemina were probably not so much interfered with by pressure as by extension of inflammation

and subsequent softening, and probably also extension of the glioma along the superior peduncle of the cerebellum into the substance of the corpora quadrigemina.

As the case progressed, it soon became evident that there was an independent affection of the cord, as evinced by the complete anæsthesia, and loss of reflex irritability in the lower extremities, as well as by the trophic changes already described.

Two suppositions could be made with regard to the affection of the cord: Either there was a tumour pressing on the cord of the same nature as that in the cerebellum, or there was extensive softening of the lumbar region. I must acknowledge that although the latter view was not free from difficulties, it was the one I adopted. In reference to the first supposition, I was only thinking of a circumscribed tumour pressing on a limited part of the cord, and this would not account for the early and total loss of reflex irritability in the lower extremities. It never entered into my calculations to think of a tumour extending the whole length of the cord. Looking back on the case with the further light which the post-mortem has thrown upon it, it seems probable that the functions of the posterior columns of the inferior dorsal and lumbar regions of the cord were interfered with by the growth of the tumour, even as early as the date of admission. This would account for the projecting forwards of the legs, and thumping of the heels, which were so like the movements of locomotor-ataxy.

*(To be continued.)*

## A CASE OF HEMIPLEGIA DUE TO WASTING OF CEREBRAL CONVOLUTIONS.

BY FRANCIS WARNER, M.D., M.R.C.P.,

*Assistant-Physician to the East London Hospital for Children,*

and

FLETCHER BEACH, M.B., M.R.C.P.,

*Medical Superintendent, Darenth Asylum.*

THIS case presented the following congenital defects, Bell's paralysis associated with disease and deformity of the right ear; congenital cyanosis, and left hemiplegia associated with destruction of certain convolutions in the right hemisphere.

The patient, Emma B——, was a girl aged 1 year 9 months, and came under observation at the East London Hospital for Children as an in-patient, July 1878. She was ill-nourished and ill-developed, exhausted, and very cyanotic, manifestly very deficient in general muscular strength, being unable to stand or sit up. She generally lay on her back, quite passive, but appeared to know her mother, and would look about her, turning her head towards any object that attracted her attention; she had not learnt to speak, but cried like other babies, and made her wants known. With the right hand she would hold and play with a toy, moving the fingers well; the voluntary movements of the right lower extremity also appeared complete. The left arm and leg remained almost constantly inactive, with only occasional very slight movement, and that purposeless and apparently quite involuntary.

On slightly pricking the left arm or leg, it was drawn up; if more strongly irritated, she tried to remove the offending pin with the right hand, but was quite unable to use the left limbs for any voluntary purpose. The left limbs were easily excited to reflex contraction, and muscular contraction to the faradic current was well marked; cutaneous sensibility appeared unimpaired. Measurement indicated no difference



in the length of the corresponding limbs; there was no difference in their circumference on the two sides, and no rigidity. The skull was not deformed, but the right external ear was very rudimentary, smaller than the left, ill-shapen, and adherent to the skull, the tragus being the only part fairly well formed; the meatus was large in its vertical diameter, and there was a depression above the meatus running inwards to a blind termination  $\frac{1}{4}$ -inch from the surface. There was much purulent discharge from the ear.<sup>1</sup> The left ear was fairly well formed, but was also the seat of otorrhœa.

The neck was very short, and its movements were limited laterally, but the head could be rotated to the due extent by passive movement. There was complete Bell's paralysis on the right side, which had existed from birth. On blowing in the child's face she winked with both eyes, but closed the right eyelids less completely. There was no paralysis of any other cranial nerve; she was readily attracted by sounds, sight was manifestly present, and the ophthalmoscope showed no abnormality; there was no difficulty in taking liquid food.

The chest was fairly well formed, and the lungs appeared healthy. The cardiac impulse was forcible, suggesting some ventricular hypertrophy; the apex beat was displaced slightly outwards; the area of cardiac dulness was well-defined, but was not perceptibly enlarged. A loud systolic bellows murmur was heard all over the precordial region, but with greatest intensity over the second left cartilage. The pulse was full and strong, cyanosis was very pronounced and constant, the fingers and toes were slightly but distinctly clubbed.

The following history was obtained from the mother. The child was born at full term. The mother while pregnant had a fright about the fourth month, but was not aware of any ill effects; at the sixth month she had a fall. The parents are both intelligent, strong, and of steady habits, and have had eleven children, all alive and healthy. At birth the deformity of the ear and neck and the facial palsy were at once noticed. Cyanosis is said to have been more marked during the first fortnight of life than later. The child could never suck, and was fed from a bottle. The weakness of the left limbs was not noted till a later period, but it appears certain that it must have existed from birth. When nine months old she was convulsed for thirty-six hours; it is said the right arm alone worked, the left remaining quiet. After this she was very exhausted for a week, and then rallied; but from this time the left arm was quite useless. No other convulsions occurred.

<sup>1</sup> For this sketch we are indebted to Mr. Basil Walker, late House Physician at the London Hospital.

The child was subsequently received into the Darent Asylum; no special symptoms occurred, and she died of bronchitis, Feb. 23rd, 1879.

Necropsy sixty hours after death. Body emaciated. Skull symmetrical. Calvaria thin, and with difficulty separated from the dura mater.

*Brain* weighed 31 ounces. The right hemisphere was smaller than the left, and after preservation in spirit for about three months, weighed  $4\frac{1}{4}$  ounces, and measured  $11.5 \times 9$  decimetres longitudinally. The pia mater was healthy at the summit of the hemisphere, slightly thickened over the frontal and occipital lobes, more thickened and absolutely diseased over an area measuring  $5 \times 4.25$  decimetres. The convolutions included in this area were the lower part of the ascending frontal and parietal, the whole of the supra-marginal, with its lobule and the superior temporo-sphenoidal. The pia mater in this region was thick and tough, its vessels very large, tortuous, and contained blood.

*Diseased convolutions.*—The ascending frontal, at a point 4.5 deci. from its summit, suddenly became shrunken to the thinness of a wafer; the nerve matter was then continued in its usual direction downwards, partially forming the roof of a large adventitious cavity in the substance of the hemisphere.

The ascending parietal convolution, at a point 6.0 deci. from its summit, suddenly underwent a similar change.

The whole of the supra-marginal, with its lobule, was shrunken and almost destroyed.

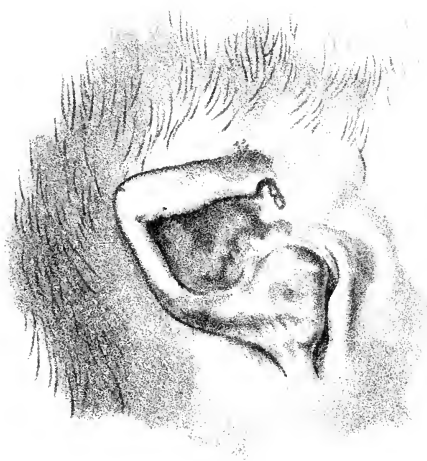
The superior temporo-sphenoidal was much wasted. On examining it, a cavity was found lying between the layers of the wasted convolution, and lined with fibrous tissue.

The adventitious cavity lying below the wasted convolutions (except the superior temporo-sphenoidal) measured  $5 \times 3$  deci.; and was also lined with thickened fibrous tissue, a band of which passed across it, forming a septum. The floor, generally smooth, with the exception of a projection the size of a hazel-nut situated at its posterior part, was composed of a very thin layer of nerve tissue forming the roof of the lateral ventricle. Neither of these cavities appeared to be a serous or blood cyst, or the sac of an abscess. They did not communicate with one another.

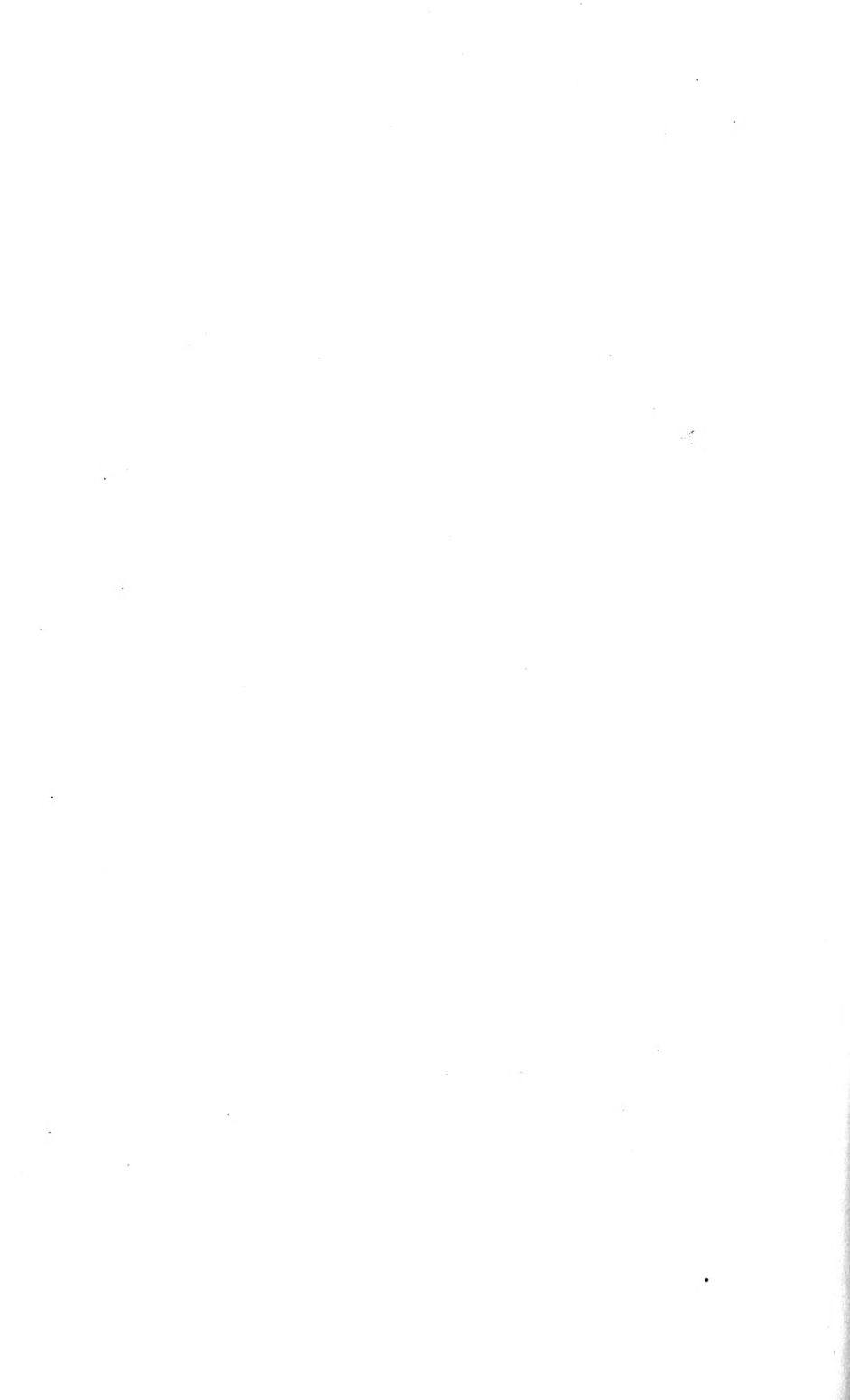
The lateral ventricle appeared normal; its ganglia and the choroid plexus were healthy.

The rest of the brain was healthy, and the convolutions were of normal size.

The head only was allowed to be examined.



Case of Hemiplegia due to Wasting of Cerebral  
Convulsions. (Warner & Beach.)



*Remarks.*—In this case it appears certain that the left hemiplegia existed at birth, and was due to intra-uterine wasting of the convolutions described, for during life there was no illness (except one attack of convulsions) which could account for the large amount of localised cerebral atrophy. The hemiplegia was characterised by total loss of voluntary power; but sensation, reflex action, electric contractility, and good nutrition remained. There was no spastic contraction or convulsion of the affected side. It may be noted that the seat of atrophy included a considerable area of Ferrier's motor regions. The congenital ear defect was obviously connected with Bell's paralysis, and it is noteworthy that it existed on the same side as the cortical lesion. The congenital cyanosis appears to lend force to the argument that the disease of the brain was of intra-uterine character.

## Abstracts of British and Foreign Journals.

**On the Sensory Centres in the Cortex Cerebri.** LUCIANI and TAMBURINI (*Studi Clinici sui Centri Sensori Corticali, Comunicazione preventiva dei Professori Luigi Luciani e Augusto Tamburini. Milan, 1879*).—These distinguished observers have endeavoured to collect cases where lesions in the brain were found in connection with deficiency of any of the senses, in the hope of throwing light upon the new localisations of sensory centres in the brain, attempted by Munk, Ferrier, and others. They commence with the announcement that Schiff has at length been induced by the clinical researches of Charcot and Pitres, and those of Maragliano, to admit the existence of such centres in man. From different authors they have collected forty-one cases of lesion connected with sensory deficiencies, thirty-four of which are failures of vision, (amblyopia, hemiopia, blindness, &c.). In three there was deficiency both of sight and hearing; in one the hearing alone was affected, and in three more the lesions were of a diversified character. The authors find themselves led by their inquiries to admit that the psycho-sensory centres of vision are situated both in the angular gyrus and in the occipital lobe. In this way they at once reconcile the rival theories of Ferrier and Munk, and get rid of negative observations. The cases collected are taken from medical writers in several European languages. Many of them, not having been composed with a view to illustrate these novel theories, are wanting in detail.

Out of thirty-four cases of deficiency of vision, the pathological lesion was in thirteen cases in the right hemisphere; in thirteen in the left; and in eight in both hemispheres. Of the thirteen instances in which the lesion was in the right hemisphere, the deficiency of vision was in five cases in the left eye; and in eight it appeared to be in both eyes. In the cases in which the lesion occupied the left side, four had deficiency in the right eye, and in nine the deficiency was on both sides, or, at least, the

loss of sight was not limited to one eye. In fourteen cases the lesion occupied one spot alone; in eight of these it was in the parietal lobe, in two of the eight it was in the angular gyrus. In six cases, it was in the occipital lobe.

In two cases of deafness there were lesions of the temporal convolutions.

The authors consider the results of their inquiry to be at least promising.

They present the following conclusions:—

1. Clinical observations confirm the results obtained in our experiments on animals, as to the localisation of the sensory zone in general and of the visual centre in particular.

2. The negative clinical cases may be regarded either as inquiries imperfectly conducted or recorded, or as lesions where the whole sensory zone was not implicated. Or perhaps the slowness with which the disease advanced allowed time for compensation, either by the function being vicariously assumed by the centre on the opposite side, or by the nasal secondary centres.

W. W. IRELAND.

**On a Case of Progressive Muscular Atrophy, with Lesions in the Anterior Horn of the Cord.** ERB and SCHULTZE (*Arch. f. Psych. u. Nerv. Kr.* ix. 369).—The publication of this case is intended as an answer to that of Lichtheim (*ibid.* viii. 1878), which the latter gave as one of *typical* progressive muscular atrophy, without lesion of the cord. Strong doubts are raised by Erb and Schultz as to the correctness of the diagnosis: first, because the clinical evidence is not in concordance with it; second, because the post-mortem appearances of the muscles showed them to be affected with simple, not with *degenerative* atrophy. The former is characterised by a mere quantitative diminution of all the constituents of muscular tissue; the latter, by proliferation of nuclei, loss of striation, waxy degeneration, &c.

In the present case the clinical characters corresponded with the classical picture of the disease. The post-mortem appearances of the muscles were both to the naked eye and to the microscope typical. Macroscopically the cord presented no change. Hardened and stained sections showed changes in the central part of the grey matter. There almost nothing but Deiter's cells, some of them much enlarged, are to be seen. The ganglionic cells of the middle group are much degenerated; those of the anterior and posterolateral groups almost all normal. Clarke's columns and all the

white matter normal. Anterior roots normal throughout. There was then no continuity between the seat of spinal and of muscular lesions, a fact which gives support to the theory that there are trophic centres for the muscles independent of those of the nerves. Now the question arises: Which is here the secondary lesion? Did the nerve-centres degenerate in consequence of the muscular atrophy, or *vice versá*? Against the former view the authors adduce a number of arguments; and without wishing to appear dogmatic, conclude that the muscular depends upon the spinal lesion.

**On Concussion of the Spinal Cord.** OBERSTEINER (*Medizinische Jahrbücher*, iii. iv. 1879, and *Separat-Abdruck*, pp. 32).—By this expression the author understands those cases where the spinal functions are altered in consequence of mechanical violence, direct or indirect, to the vertebral column, there being no anatomical lesion of the cord to account for such alterations. There is a “chronic” form of concussion, hitherto little studied, due to a series of minute shocks, which the author has observed among those employed on the tramways, and which manifests itself as weakness and unsteadiness of the legs. The ordinary form of concussion may be due to (1) falls or blows, (2) gunshot injuries, (3) railway accidents.

The author describes the case of a soldier who was shot in the nape of the neck, the bullet lodging near the third dorsal vertebra, without injuring the bony canal. He immediately felt intense pain in the legs, but could not move them. Three weeks after he had complete paraplegia and anæsthesia as high as the umbilicus, paralysis of the bladder with incontinence of fæces, extensive decubitus, &c. A careful post-mortem examination was made, which revealed extensive peritonitis and inflammation of the urinary organs. The middle third of the dorsal cord was diffuent, and the meninges there thickened and inflamed. Above and below, the large cells of the anterior grey matter presented alterations in the shape of vacuoles, or colloid drops in the protoplasmic constituents, alteration in the shape of the nucleus. Other cells were studded with numerous particles, which readily took the carmine stain. The other constituents of the cord, and the nervous roots, also showed degenerative changes. Below the seat of the main lesion the lateral columns were degenerated, and more or less extensive softened portions of the cord, with loss of substance, were noticed. Above, the posterior columns, together with the posterior



peripheral portion of the lateral columns, and Flechsig's "lateral fasciculi from the cerebellum" showed signs of ascending degeneration, which could be followed, in the case of the columns of Goll, into the medulla, in the case of the lateral columns into the corpora restiformia.

Discussing the symptoms of concussion, the author points out that it is necessary to distinguish between general and partial concussion; and also between the cases where the effects are immediately felt, and those where the symptoms are insidiously developed in time, as happened in 43 per cent. of the cases he tabulates. The prognosis is far more serious in the latter.

The leading symptoms are paralysis and anæsthesia of the extremities, and paralysis of the bladder and rectum, to which numerous others associate themselves differently in different cases.

As a rule, the pathological process, apart from secondary degenerations, is diffuse, and not limited to any system of fibres or cells. Tumours occasionally owe their origin to concussion.

The phenomena of concussion have been sometimes ascribed to vaso-motor effects; but the author refutes this theory, and believes that they are due to molecular changes in the nervous elements of the cord, whose immediate effects may vary, and which subsequently may undergo regression, or lead on to myelitic processes. Obersteiner brings his paper to a close with some remarks on secondary degenerations generally, showing the importance of the view that all the fibres undergoing ascending degeneration are centripetal to the cerebellum only. With reference to their pathogeny, he says that the blood-vessels are not primarily implicated. Nor does he agree with Erb in the viewing these secondary degenerations as "trophic" changes. He believes they originate in the lymphatic spaces which convey, upwards or downwards, the products of the myelitic softening at the point of lesion.

**On Localisation of Atrophic Spinal Paralyses.** REMAK (*Archiv für Psychiatrie*, ix. 3. *Separat-Abdruck*, pp. 126).—In a previous paper (*ibid*, vi. 1) the author had discussed the probable spinal origin of lead palsy, and pointed out the great similarity between it in its generalised form and the anterior poliomyelitis of the adult. He had also shown that the exemption of the supinator longus in saturnine wrist-drop also occurred in non-toxic forms of spinal disease. Other muscles, such as the tibialis anticus and sartorius, are frequently found to remain intact, whilst all the other muscles supplied by the same nerve undergo atrophy. It is

to be remarked that electro-diagnosis gives us no direct clue to the spinal localisation of the original disease, but simply to the distribution of the atrophic process among the muscles; the former is to be inferred by reasoning from the latter. Again, it is evident that disease of the anterior roots will present the same clinical picture, as disease of the spinal centre of these roots themselves. Such is the case, for instance, in lesions of the fifth and sixth roots of the cervical plexus.

From a purely physiological point of view it is probable that various simple forms of movements depending upon co-ordinated action of several muscles, depend upon centralised mechanisms in the cord. But it is upon clinical evidence that we must mainly rely for the present in localising disease of the anterior horn of grey matter.

In the present paper Remak adduces numerous observations of his own, which he fully describes and minutely discusses, and shows how, so far, the results of autopsies concord with his results. Of these, we must content ourselves here to give but a bare outline. With reference to the spinal paralyses of the arm, he distinguishes an upper arm and a forearm type.

In the first the deltoid, biceps, brachialis anticus, and supinator longus, are affected together. When one of these muscles is seriously impaired, traces at least of nutritive changes are always to be discovered in the others by electrical investigation. But they frequently are visibly affected all at once (e.g. in cases of poliomyelitis, infantile or adult); and a case of lead palsy is adduced, in which this was the case. This state of things curiously coincides with that observed in some cases of lesion of the brachial plexus (Erb, Remak, Hoedemaker); and it must be remembered that the muscles just mentioned are those made to contract by exciting the fifth and sixth roots of the cervical plexus, at the supraclavicular point. Their spinal centre is situated high up in the cervical enlargement; and this explains the immunity of the supinator among the forearm-muscles, supplied by the musculospiral in cases of lead palsy and the like.

In the well-known forearm type, the muscles implicated are the extensors of the fingers and wrist. Their spinal centre is probably in the middle of the cervical enlargement. The extensor ossis metacarpi pollicis, seems to be centrally connected more with the inter-ossei, short-thumb, and flexor muscles of the hand. Their centre would be in the lower portion of the enlargement; and this assumption is supported by direct anatomical proof.

What precedes is based mainly upon observation in atrophic paralyses. The distribution of progressive muscular atrophy does certainly not present such clear types; but in many instances is in sufficiently close conformity to them, to indicate the spinal pathogeny of the disease.

With reference to the thigh, in the domain of the crural nerve, we find the sartorius enjoy considerable immunity. On the other hand, Remak mentions a case where it was found implicated along with the ilio-psoas. [This fact, taken in connection with the anatomical configuration of the lumbar plexus, seems to justify the assumption that the flexors of the thigh, of which the sartorius is one, are supplied by the second lumbar nerve.] The nucleus of the tibialis anticus is near that of the vasti and rectus femoris, with which it is often found to suffer. At the same time it must enjoy a certain independence, since this muscle is frequently found alone implicated; and, on the other hand, it is rarely affected along with the peronei and extensor muscles of the toes. [This would point to the fourth lumbar, as containing the fibres to the tibialis anticus.] In lead paralysis of the leg it has never been found paralysed, thus resembling the supinator in the arm. Autopsies point out the mid-lumbar enlargement as containing its motor nucleus.

The muscles of the shoulder-girdle and pelvis do not yet admit of spinal localisation. No leg types can yet be framed, as paralysis, though most frequently implicating the extensors, not infrequently extends to the flexors in various combinations.

Lesions of plexus roots imitate the distribution of poliomyelitic disease, from which other circumstances, such as sensory troubles, must help to distinguish them.

A. DE WATTEVILLE.

**On the Structure of the Cortex Cerebri.** STRICKER and UNGER. At the meeting of the Acad. der Wissensch. of Vienna, on July 10, 1879, Prof. Stricker and Dr. L. Unger brought forward the results of a research on the structure of the cortex. The following are the main conclusions:—

(1) In the cortex there is a single fine network, composed of fibres both of nervous and connective tissue.

(2) This network is composed partly of the processes of the ganglion cells, or their axial prolongations, and partly of the processes of connective tissue-corpuscles.

(3) Every variety of transitional form between connective tissue-corpuscles and proper nerve cells is found.

(4) The meshes of the network are filled with a fine granular or homogeneous substance.

(5) This network, with its substance, is analogous to the ground-substance of bone, cornea, and other connective tissues.

**Shrivelling and Atrophy of the Cornu Ammonis in Epilepsy.**—Dr. L. Pfleger, of Vienna (*Allg. Zeitsch. f. Psychiatrie*, Bd. 36), has had opportunities of post-mortem examination in a large number of epileptics—43 in all—and finds that in somewhat over the half of the cases, one or both cornua are shrivelled or atrophied; the unilateral degeneration being once again more common than bilateral. In his cases the degeneration was especially common in women, but this difference does not exist when all the recorded cases are taken together. From his table it also appears that in unilateral shrivelling the left is more commonly affected in males; whereas in females it is the right; in both cases in the proportion of 7:15.

**A Case of Cortical Epilepsy.**—Prof. Drasche (*Wien. Med. Wochensh.* No. 39, 1879) records the following case. A tubercular patient, aged 24, observed on July 14 a powerful contraction in his left wrist, and rigidity of the fingers. This passed off, but recurred on attempts at using the hand. This was soon followed by spasms of the fingers of the left hand. On the 18th the left side of the face, and both limbs (especially the upper) on the left side became parietic. Slight sopor. On the 19th instant epileptiform convulsion, with loss of consciousness; the convulsions affecting the left side. This was followed by complete left hemiplegia. On the 21st two similar attacks. Coma became deeper, and death on July 28.

*Section.*—Old standing tubercle of lungs. Chronic tubercular meningitis on the convexity of right hemisphere. On the anterior and posterior central convolutions (ascending frontal and parietal), several tubercles of the size of peas, invading the cortex, and surrounded by softening. Acute basilar meningitis.

H. OBERSTEINER.

**On the Pathological Anatomy of Hydrophobia.** WELLER (*Arch. f. Psych.* ix. p. 493).—The following conclusions are founded upon an examination of the nervous systems of seven dogs and one man, death in all cases having resulted from hydrophobia. (1) Hydrophobia shows itself in the nerve-centres as an inflammatory process, originating in the vessels. It is characterised by

perivascular exudation, and infiltration of the tissues with lymphoid elements; the leucocytes are aggregated, more or less closely, in clusters or foci. At the same time, and probably as a product of degenerative changes in the nerve elements, peculiar fatty bodies appear in the perivascular spaces. These fatty bodies are pathognomic of hydrophobia: in no other disease have objects resembling them in size, form, and number hitherto been observed. They are identical with the "highly refractile hyaloid substance" of earlier authors, and are best seen in fresh preparations. (2) The inflammatory process is an early stage of acute myelitis or encephalitis. That it does not, like acute myelitis generally, go on to softening, depends on the short duration of the disease, this again being probably due to the localisation of the lesion. (3) The pathological changes were most marked in the medulla oblongata, especially in the nuclei of the eighth nerve. In the spinal cord, the parts most affected were the surroundings of the central canal, the anterior cornua, and the margins of the posterior cornua. (4) In the human brain, the process was confined to the spinal cord and medulla; in the dogs' brains, it was seen also in the corpora quadrigemina, the basal ganglia, and the cerebral hemispheres; it was frequently found in the olfactory convolutions, but very rarely in any other part of the hemispheres, and in the cerebellum it was never found.

### On Combined (Primary) Disease of the Spinal Tracts.—

WESTPHAL contributes a lengthy paper on this subject to the *Archiv f. Psychiatrie* (viii. p. 469, ix. pp. 413 and 691). Five cases are reported: in four of them there was lesion of the posterior and lateral columns through the whole length of the spinal cord, the lesion in one instance extending to the anterior columns and the anterior cornua; in the fifth case (Schneider's) the lateral columns were implicated throughout their extent, but the posterior columns were unaffected in the lumbar region. These cases are considered respectively from an anatomical, pathological, and clinical standpoint.

The lesion of the lateral columns was in no case *systematic*; it was never confined to any of the systems of fibres into which Flechsig divides these columns. Whether the affection of the posterior columns was systematic is doubtful, and cannot be decided until we know more of the anatomy of this part of the cord. Fibres of different systems were, however, not unfrequently found affected in a portion of their course. Various attempts have been made to explain the localisation of the lesion. Some have

thought that the disease spreads directly from posterior to lateral column; but Westphal shows, by a careful examination of his own and other cases, that the lesions of the two columns are in a manner independent, and that the pathological process certainly does not extend directly from one to the other. Others have held that the initial lesion is a chronic inflammation of the posterior columns, which sets up a meningitis posterior; this again, by its extension, carrying the process to the lateral and anterior columns. A third view is that the lesion is simply a chronic myelitis, which, for some unknown reason, spreads chiefly in a longitudinal direction. This view, though beset with difficulties, is provisionally accepted by Westphal. Whether the lesion originates in any special focus or simultaneously affects the whole length of the strands is not known, but it would appear that the upper two-thirds of the dorsal part of the cord are specially concerned in its origin, for here the disease is often first observed and most severe. Westphal suggests that the peculiar distribution of the lesion may be due to the primary implication of the nerve-cells of the grey matter, for these are connected with the fibres of different systems. The author remarks that the lateral limiting layer of Flechsig (*seitliche Grenzschicht der grauen Substanz*) is always left intact.

Pathologically, the white matter showed two varieties of disease: (1) granular degeneration, or chronic myelitis; (2) grey degeneration, or sclerosis. The former, with one exception, was always found in the lateral columns, the latter always in the posterior columns. It must not be concluded, however, that the posterior columns never show the granular degeneration, for not unfrequently it occurs in quite typical cases of *tabes dorsalis*. But if the posterior columns exhibit granular degeneration, the lateral columns also invariably exhibit the same degeneration; grey degeneration of the lateral (or anterior) columns never co-exists with granular degeneration of the posterior columns. The relations of the two varieties have often been discussed under this form. Is the secondary degeneration of Türk (a granular degeneration) essentially the same as sclerosis (grey degeneration). Westphal holds that it is. He shows that the two are connected by intermediate stages, and sometimes are associated together; for instance, in typical cases of grey degeneration the sclerosed parts are not seldom surrounded by a small zone with all the characters of granular degeneration, from which it would appear that the granular is but the earlier stage of the grey degeneration.

Clinically, it is of interest to inquire in what way the symptoms

proper to lesion of the posterior columns are modified by lesion of the lateral columns. From his own four observations and from two others, Westphal concludes that where the lateral columns, and especially their pyramidal strands, are affected, the symptoms of ataxia are accompanied or replaced by those of paralytic weakness. This conclusion would seem to be stultified by a case reported by Friedreich, in which, though the lateral and anterior as well as the posterior columns were diseased, the muscular power remained intact. This case certainly shows that a considerable part even of the pyramidal strands may be affected without any symptoms of motor weakness, but is not conclusive against the position just taken, for the degenerative changes were not sufficiently intense to make the case a crucial one; e. g. in the inferior parts of the cord they were not very marked, and they were less advanced in the lateral than in the posterior columns. Westphal next discusses the view that the motor weakness in these cases is due to lesion of the posterior columns themselves. Leyden has published a case in which there was ataxia and great motor weakness of the lower extremities, and yet the posterior columns alone were degenerated. Leyden thinks that in this and similar cases the motor weakness is a pseudo-paralysis, and is to be attributed not to any particular anatomical lesion, but to a want of innervation or force of will. He finds support for his view in this fact, among others, that ataxic women present the symptom much oftener than men. Westphal does not accept this view. He points out how inapplicable it is to cases of one-sided paralytic weakness. This solitary case of Leyden does not, in his opinion, preclude us from holding that in the numerous cases of ataxia with motor weakness and lesion of the lateral or anterior columns, the lesion and symptom are connected as cause and effect. Lastly, Westphal discusses the theory that the motor weakness is caused by changes in the muscles themselves. His conclusion is that neither simple atrophy nor atrophy with degeneration satisfactorily explains the impaired motility. The author sums up this part of his paper by stating that the motor weakness in these cases is most probably produced by the lesion of the lateral or anterior columns, though, as yet, the more precise relation of the symptoms to the localisation of the lesion remains unknown.

Westphal's cases lend no support to Charcot's statement, that where there is lesion of the lateral columns, the paresis or paralysis is always accompanied by contracture. They show, on the contrary, that in combined disease of the posterior and lateral columns

muscular rigidity and spastic contracture are not observed, if the lesion of the posterior columns extends to the lumbar portion of the cord and affects the parts known as the root-zones. In three of the cases symptoms of affection of the trigeminus were observed, viz. paræsthesia, painful sensations in skin, pain in chewing, analgesia, lachrymation, and (in one case only) neuroparalytic keratitis. These disturbances of sensibility were associated with certain peculiarities of physiognomy, which are to be regarded as evidence of an ataxia of the facial muscles. The trigeminus presented the same pathological changes as the posterior nerve-roots; the atrophy of the ascending root could be traced into the medulla oblongata. Fits of reddening of the face, salivation, perspiration, dyspnoea, and enormously heightened rapidity of pulse were several times noticed, these symptoms being doubtless due to some bulbar lesion. In one case, where the sensibility of the skin was almost normal, and that of the muscles very considerably affected, the lesion of the posterior columns was localised in a peculiar manner; a strip of white matter bordering the posterior septum, the posterior commissure, and the posterior cornu was left intact. This case is interesting in connection with the supposed different courses taken by the sensory fibres of skin and muscle.

The diagnosis of combined disease of the posterior and lateral (or anterior) columns rests mainly on two symptoms; (1) the peculiar gait, which differs from that of an ordinary ataxic; and (2) the appearance of muscular weakness, with or without evident ataxia. But these symptoms are not pathognomonic; they may be present when the posterior columns alone are diseased.

Schneider's case presents several points of interest. There was pseudo-secondary degeneration of the posterior columns, and of the pyramidal and cerebellar tracts of the lateral columns; that is to say, though the lesion was a primary and independent one, it resembled a secondary degeneration in its distribution. At the posterior part of the cord there was a chronic leptomeningitis, to which perhaps the disease of the subjacent white matter owed its origin. Diabetes complicated the later history of the case; it was brought on apparently by faradisation of the limbs, but whether due to this or to disease of the cord is uncertain. Spastic spinal paralysis was observed. It differed from the typical form described by Erb in this, that there were disturbances of sensibility and slight affection of the bladder and sphincter ani, but it does not on this account cease to be spastic paralysis. Westphal concludes his paper by some remarks on this affection, pointing out that in



cases of combined primary disease of the spinal cord it may be associated with (1) pseudo-secondary degeneration originating in the dorsal region of the cord; (2) lesion of the lateral and posterior columns, if the latter remain unaffected in their lumbar portion; and (3) disseminated sclerosis of the spinal cord or medulla (Charcot).

W. J. DODDS, M.D., D.Sc.

**Head Measurements.** DR. J. S. WIGHT, of Brooklyn, U.S. (*Archives of Medicine*, vol. ii. No. 2, Oct. 1879, New York), in an elaborate paper, gives the results of observations made on the heads of 21 males and 21 females (uneducated), and 21 males and 21 females (educated). The subjects were all adult.

His paper tends to show that, in both educated and uneducated, the brain of the male has a comparatively greater volume in the *anterior* part of the cranial cavity than the brain of the female, whilst the female brain has a comparatively greater volume in the *superior* part of the cranial cavity than that of the male; and the education in both sexes tends to increase the volume of the anterior part of the brain. He demonstrates that the volume and form of the brain of the uneducated male somewhat nearly resemble the volume and form of the brain of the educated female; while in relation to education the female shows greater variation, in the development of the anterior part of the brain, than the male.

He considers that the female brain shows as great a capacity for development by education as the male brain, and under similar circumstances of mental work and heredity would be fully equal to it.

He argues from this that females ought to receive a higher education, which he thinks would tend to the amelioration of the race. He believes his conclusions will be supported by the facts of imperfect development operating as causes of diseased conditions.

In making his measurements Dr. Wight uses a somewhat indefinite spot on each mastoid process as a centre of departure; and from one mastoid centre-point to the other measures five transverse arches, which he calls inter-mastoid arches.

- (1) An anterior arch above supra-orbital ridges.
- (2) A frontal arch over frontal eminences.
- (3) A middle arch nearly over coronal suture.
- (4) A superior arch nearly over vertex, and about parallel with axis of foramen magnum.
- (5) A posterior arch over the occipital protuberance.

Three diameters :—

- (1) A base line from one mastoid point to the other.
- (2) The greatest transverse diameter.
- (3) The greatest antero-posterior diameter.

He also takes the greatest antero-posterior circumference (just above frontal sinuses and occipital protuberance).

He makes a great point of the masto-frontal angle, which is included between the planes of the anterior and superior arches, and the size of which, he says, gives an important indication of the development of the brain.

The greater the masto-frontal angle, *cæteris paribus*, the greater the volume of brain in the anterior part of cranial cavity.

This masto-frontal angle is apparently included between the fronto-mastoid line and the base-line of the skull, though formation is not clearly described in the paper.

CROCHLEY CLAPHAM.

**General Paralysis amongst Negroes.**—The American *Journal of Insanity* for October 1879, records in the "Proceedings of the Association of Medical Superintendents" some highly interesting remarks on this subject. Dr. Bryce, of Alabama, stated that notwithstanding a large experience amongst insane negroes, he had never met with a case of general paralysis amongst them. Dr. Callender, of Tennessee, had no recollection of having seen in the African any type of disease resembling general paralysis of the insane; and Dr. Nichols, of Washington, said that having begun in 1855 to have the supervision of twenty coloured patients—a number now increased to fifty—he had never in that experience seen a case of general paresis. It was stated that Drs. Compton, Longworthy, and Wallace, had also affirmed that they had never seen a case of paresis amongst the coloured people of the South. Dr. Black, quoting from the last report of the Central Asylum for the Coloured Insane in Virginia, stated that four cases had been admitted into that institution previous to last year. It is evident that general paralysis is characteristically rare amongst negroes.

ROBERT LAWSON, M.B.

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